A. M. A.
ARCHIVES OF
OTOLARYNGOLOGY

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

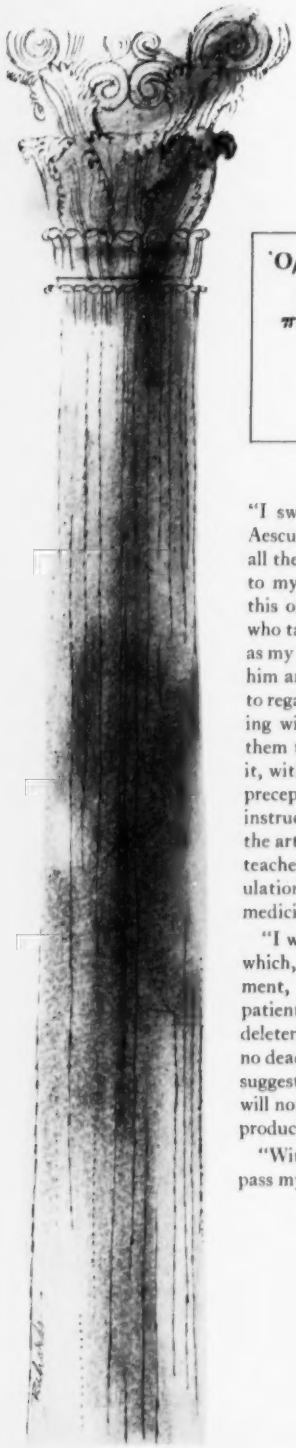
JOHN D. KERNAN, New York
RALPH A. FENTON, Portland, Ore.
ERNEST M. SEYDELL, Wichita, Kan.
CHEVALIER JACKSON, Philadelphia
ROBERT L. GOODALE, Boston
J. MACKENZIE BROWN, Los Angeles
CARL H. McCASKEY, Indianapolis

JUNE 1954
VOLUME 59 NUMBER 6

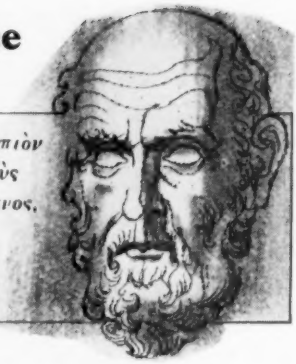
Published Monthly by
AMERICAN MEDICAL ASSOCIATION
535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of March 3, 1879. Annual Subscription, $12.00

TABLE OF CONTENTS FIRST PAGE



Tradition of service



Ὀμνύω Ἀπόλλωνα ἰητρὸν καὶ Ἀσκληπιὸν
καὶ Ὑγείαν καὶ Πανάκειαν καὶ θεοὺς
πάντας τε καὶ πάσας, ἵστορας ποιεύμενος,
ἐπιτελέα ποιήσειν κατὰ
δύναμιν καὶ κρίσιν ἐμὴν ὅρκον
τόνδε καὶ συγγραφὴν τήνδε·

"I swear by Apollo, the physician, and Aesculapius, and Health, and All-heal, and all the gods and goddesses, that, according to my ability and judgment, I will keep this oath and stipulation: to reckon him who taught me this art equally dear to me as my parents, to share my substance with him and relieve his necessities if required; to regard his offspring as on the same footing with my own brothers, and to teach them this art if they should wish to learn it, without fee or stipulation, and that by precept, lecture and every other mode of instruction, I will impart a knowledge of the art to my own sons and to those of my teachers, and to disciples bound by a stipulation and oath, according to the law of medicine, but to none others.

"I will follow that method of treatment which, according to my ability and judgment, I consider for the benefit of my patients, and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to anyone if asked nor suggest any such counsel; furthermore, I will not give to a woman an instrument to produce abortion.

"With purity and with holiness I will pass my life and practice my art. I will not cut a person who is suffering with a stone, but will leave this to be done by practitioners of this work. Into whatever houses I enter I will go into them for the benefit of the sick and will abstain from every voluntary act of mischief and corruption; and further from the seduction of females or males, bond or free.

"Whatever, in connection with my professional practice, or not in connection with it, I may see or hear in the lives of men which ought not to be spoken abroad, I will not divulge, as reckoning that all such should be kept secret.

"While I continue to keep this oath unviolated, may it be granted to me to enjoy life and the practice of the art, respected by all men at all times, but should I trespass and violate this oath, may the reverse be my lot."

SONOTONE, an organization dedicated to the service of the hard of hearing, reprints the Hippocratic Oath in full as a salute to humanity's greatest public servant, the doctor.

All SONOTONE Hearing Aids are on the list of A. M. A. Council accepted devices.

Sonotone Corporation
Elmsford, N. Y.

TABLE OF CONTENTS

ORIGINAL ARTICLES

A New Treatment for Chronic Secretory Otitis Media PAGE

B. W. Armstrong, M.D., Charlotte, N. C. 653

Treatment of Nasal Polypi, with Comments on Frontal Sinus Surgery

J. A. Harpman, M.S., B.Sc., M.B. (London), F.R.C.S. (Edinb.), D.L.O. (Eng.), Warwickshire, England 655

Exogenous Foreign Bodies of the External Auditory Canal, Nasal Cavity, and Esophagus

J. C. Peele, M.D., Kinston, N. C. 672

Modern Aspects of the Recruitment Phenomenon

Martin P. Lansberg, M.D., Soesterberg, Netherlands 712

Circulation of the Spiral Ligament and Stria Vascularis of Living Guinea Pig

Francis L. Weille, M.D.; Samuel R. Gargano, B. S.; Raymond Pfister, M.D.; Daniel Martinez, M.D., and John W. Irwin, M.D., Boston 731

Rhinoplastic Applications to Submucous Resection of the Nasal Septum

Alan L. Rowland, M.D., San Diego, Calif. 739

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Adenoidectomy with Hemostasis

Max Unger, M.D., New York 744

Atrophic Rhinitis

Richard L. Ruggles, M.D., Cleveland 747

PROGRESS IN OTOLARYNGOLOGY

Allergy of the Eye, Ear, Nose, and Throat

Ernest L. MacQuiddy, M.D., Omaha, and John M. Sheldon, M.D., Ann Arbor, Mich. ... 749

SPECIAL REPORTS

Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation of the American Medical Association, as of May 1, 1954 763

REGULAR DEPARTMENTS

Abstracts from Current Literature 767

Books 780

Directory of Otolaryngologic Societies 781

Notice

THE INDEX FOR THIS VOLUME WILL BE PUBLISHED AS SECTION II OF THE JULY ISSUE.

AMERICAN MEDICAL ASSOCIATION
A. M. A. Specialty Journals

Instructions to Contributors

Communications regarding editorial management, subscriptions, reprints, etc., should be addressed to Specialty Journals, American Medical Association, 535 North Dearborn Street, Chicago 10.

Articles, book reviews, and other materials for publication should be addressed to the Chief Editor of the Specialty Journal concerned. Articles are accepted for publication on condition that they are contributed solely to that journal.

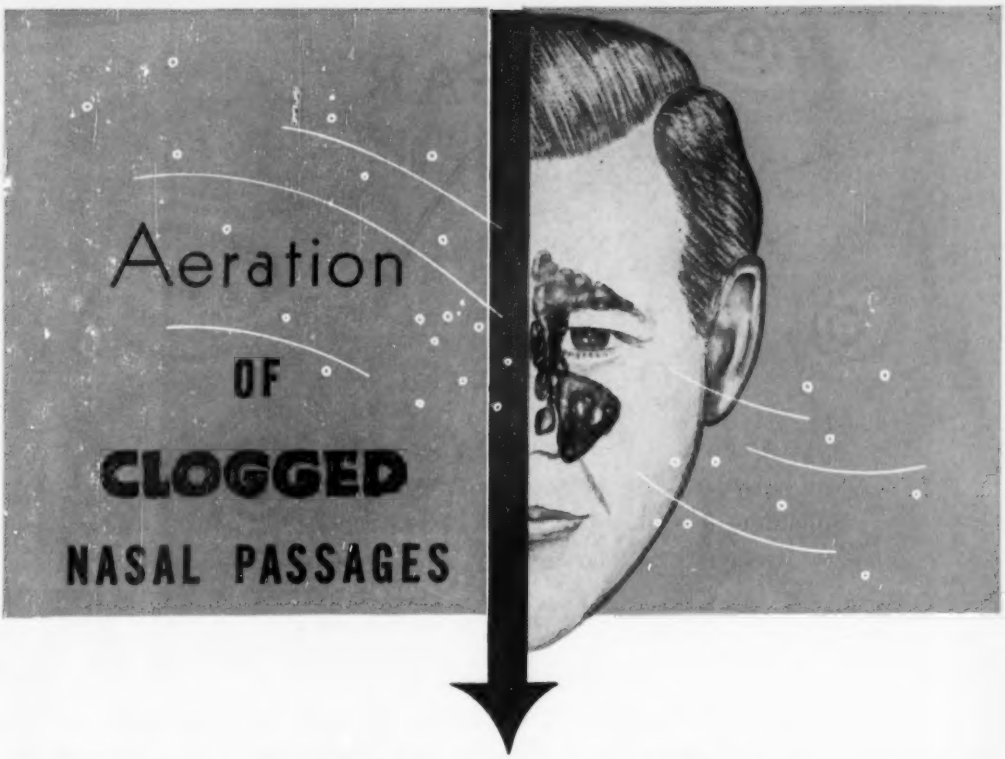
An original typescript and the first carbon of an article should be provided, and must be double or triple spaced on one side of a standard size page, with at least 1-inch margin at each edge. An article in English by a foreign author should be accompanied by a draft in the author's mother tongue. Improvised abbreviations should be avoided.

The maximum illustration space on a journal page is 5 by 8 inches. Original line drawings should not exceed 8½ by 11 inches. Oversized originals should be photographed and a print submitted within the 5 by 8 limits. Prints larger than 5 by 8 inches will be reduced in scale and/or cropped. Photomicrographs larger than 5 by 8 inches will be reduced in scale unless portions to be cropped are indicated by the author. Any cut-off marks should be made on the margins or mountings rather than on the illustration itself. Charts and drawings should be in black ink on hard, white paper. Lettering must be large enough to permit necessary reduction. Glossy prints of x-rays are requested. Paper clips should not be used on prints, since their mark shows in reproduction, as does writing on the back of prints with hard lead pencil or stiff pen. Labels should be prepared and pasted to the back of each illustration showing its number, the authors name, an abbreviated title of the article, and top plainly indicated. Photographs should not be rolled unless too large to be sent flat. If mailed flat, protect them with several layers of corrugated board. A rolled photograph should face outward. Charts and illustrations must have descriptive legends, grouped on a separate sheet. Tables must have captions.

References to the literature should be numbered in the order in which they are referred to in the text or listed in alphabetical order without numbers. A chronological arrangement, with all entries for a given year alphabetized according to the surname of the first author, may be used if preferred. References should be typed on a special page at end of manuscript. They should conform to the style of the *Quarterly Cumulative Index Medicus*, and must include, in the order given, name of author, title of article (with subtitle), name of periodical, with volume, page, month—day of month if weekly or biweekly—and year. Names of periodicals should be given in full or abbreviated exactly as in the *Quarterly Cumulative Index Medicus*. Reference to books must contain, in the order given, name of author, title of book, city of publication, name of publisher, and year of publication. Titles of foreign articles, if originally in a generally known Romance or Germanic tongue, must either all be in English translation, preferably that used in the *Quarterly Cumulative Index Medicus* subject entries, or all in the original language. Titles in other languages must be translated. The author must assume responsibility for the accuracy of foreign titles.

Matter appearing in the A. M. A. Specialty Journals is covered by copyright, but as a rule no objection will be made to its reproduction in a reputable medical journal if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the A. M. A. Specialty Journals, or in any other publications issued by the Association, will not be permitted.

AMERICAN MEDICAL ASSOCIATION
535 North Dearborn Street Chicago 10



Breathing comfort as well as proper drainage and aeration of the sinuses during upper respiratory infections is assured by the swift and prolonged decongestive action of

NEO-SYNEPHRINE®

HYDROCHLORIDE

By shrinking the swollen mucosa, Neo-Synephrine permits drainage of purulent matter, restoring free breathing and relieving the headache caused by clogged passages.

RAPID AND PROLONGED ACTION — Clearing of nasal obstruction follows within seconds after application of Neo-Synephrine and is unusually prolonged, so that comparatively few daily applications are necessary throughout the course of a cold.

WELL TOLERATED — Neo-Synephrine is notable for its relative freedom from sting, virtual absence of compensatory congestion and also has been found relatively free from systemic side effects such as nervous excitation, cardiac reaction or insomnia even when tested on hypertensive, cardiac and hyperthyroid patients.[1]

NO APPRECIABLE INTERFERENCE WITH CILIARY ACTIVITY — Neo-Synephrine not only restores nasal patency, but is compatible with ciliary action.

NO DROWSINESS — Neo-Synephrine may be used by the ambulatory patient without danger of producing drowsiness or related sedative action. Applied topically, Neo-Synephrine confines its action to the upper respiratory passages.

SUPPLIED:

0.25% solution (plain), 1 oz., 4 oz. and 16 oz. bottles.

0.25% solution (aromatic), 1 oz. and 16 oz. bottles.

0.5% solution, 1 oz. bottles.

1% solution, 1 oz., 4 oz. and 16 oz. bottles.

0.5% water soluble jelly, ⅛ oz. tubes.

●

1. Van Alyea, O. E., and Donnelly, Allen: *Arch. Otolaryng.*, 49:234, Feb., 1949.

●

Neo-Synephrine, trademark reg. U.S. & Canada, brand of phenylephrine

WINTHROP-STEARNS INC. • New York 18, N. Y. • Windsor, Ont.

ISO-PAR
(Coparaffinate)
OINTMENT

N.N.R. (1952: 44)

Indicated in the Treatment of

OTITIS EXTERNA

Archives of Otolaryngology, Sept. 1952: 241-249
The Eye, Ear, Nose & Throat Monthly, April, 1950: 200-203
Bulletin of the Johns Hopkins Hospital, Sept., 1948: 225-228
Archives of Otolaryngology, March, 1947: 294-297

Supplied in 1 lb., 4 oz., 1 oz. and ½ oz. jars

Samples and literature on request.

406 Water Street **MEDICAL CHEMICALS, Inc.** Baltimore 2, Md.

"ANNOUNCEMENT"

THE DEPARTMENT OF OTOLARYNGOLOGY

of

The Johns Hopkins Medical School

will offer a course on the

Introduction to Fundamentals of Reconstructive Surgery of the Nasal Septum and the External Nasal Pyramid

•

September 25, to October 2, 1953 Inclusive

•

Given by

DR. MAURICE H. COTTLE
Chicago Medical School

with the assistance of a faculty composed of members of The Johns Hopkins staff and invited guest teachers.

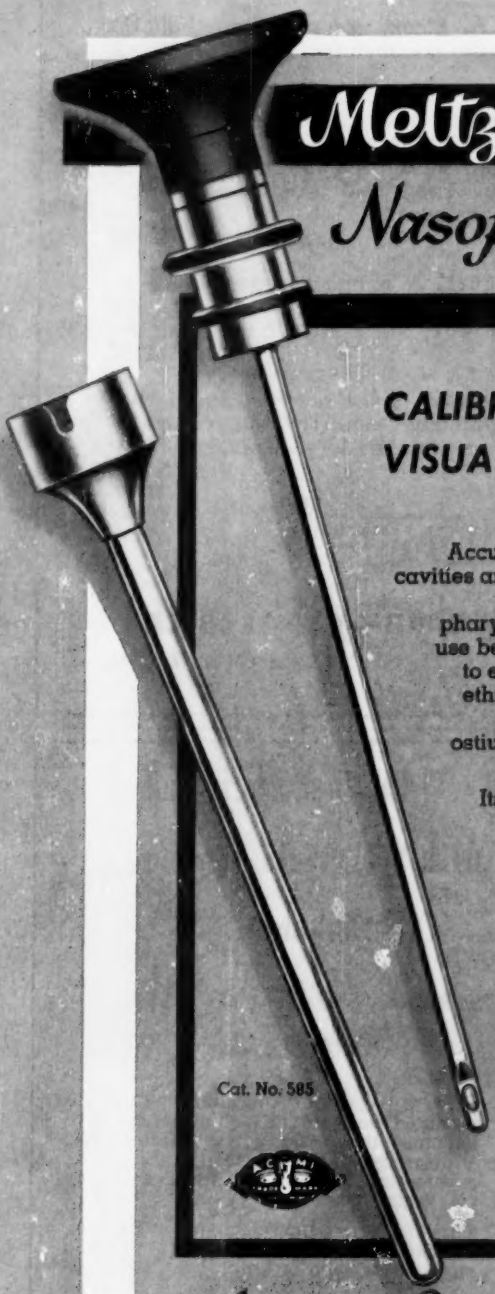
Joseph Nasal Brace for Postoperative Correction. Leather covered. Heavy Padding assures greatest comfort for patient. Including strap with metal end and jiffy buckle. Chrome plated.—

768/ST	Stand. Size, Blade length 1⅝"	$4.00
769/L	Large Size, Blade length 1⅞"	5.00
732/S	Ash Septum Straightening Forceps, stainless.....	7.50
733/S	Walsham Elevating Forceps for Septum, stainless	20.00
733/SLA	Walsham Elevating Forceps, Lateral, one blade concave, stainless	20.00

Complete RHINOPLASTY Catalog on Request

B. J. FLORSHEIM
2067 Broadway, at 72nd St., New York 23, N. Y.

Meltzer Nasopharyngoscope



CALIBRE—7 Fr.

VISUAL FIELD—large, sharply defined, high contrast

Accurate, detailed inspection of the smallest cavities and canals of the nasopharyngeal region is now possible with the Meltzer Nasopharyngoscope. Its minute calibre permits its use between the middle turbinate and septum to explore the superior meatus and sphenoethmoidal regions. It can be used under the middle turbinate to study the accessory ostium and the bulla ethmoidalis and openings of the ethmoidal cells can be seen.

Its coated lens system and cartridge type lamp produce a brilliantly illuminated, sharply defined image. The superior optical properties characteristic of A.C.M.I. telescopes are retained in the Meltzer Nasopharyngoscope. The cartridge type lamp located adjacent to the objective lens assures maximum illumination. Coated lens system provides a large visual field, sharply defined and free from internal reflections. A protective sheath that may be used as a sterilizing chamber is supplied with the instrument.

Cat. No. 585

American Cystoscope Makers, Inc.

1241 LAFAYETTE AVE. FREDERICK J. WALLACE, President NEW YORK 59, N. Y.

1

1. SMR EAR SPECULA. Black, non-reflecting, light in weight, warm to the touch, non-metallic, can be sterilized. Price **$2.50** per dozen Rack **$2.00**

2

2. LIGHT SHIELD. Consists of a polished bakelite shield to enclose an ordinary 100 watt lamp. This shield can be attached to any standard socket and the opening can be turned in any direction. The shield avoids danger from bursting bulbs and serious burns.
Price **$5.00** shield Condenser **$3.50**

3

3. SMR EXTENSION REEL. A compact method of carrying light cord. Serves the double purpose of a reel and lamp base. Contains 10 ft. of wire; wt.: 8 oz.; dia.: 3¾ in.; ht.: 1½ in. **Price $2.30**

4

4. MALLEABLE CANNULAS. These are furnished in two sizes and can be bent into any shape. The proximal end is tapered so that it can be attached to your air cut-off for drying out an ear, etc. Price **$1.25**

SMR

Surgical Mechanical Research

1905 Beverly Blvd. **Los Angeles 4, Calif.**

MANHATTAN EYE, EAR AND THROAT HOSPITAL

Announces a five-month supplementary clinical course for graduate registered nurses in the nursing care and treatment of diseases of the eye, ear, nose and throat. Operating room training is included in the course.

During the entire period the student will receive a monthly stipend of eighty dollars ($80.00) and full maintenance.

A pamphlet detailing more complete information will be sent upon request.

Apply to Director of Nursing Service
210 East 64 Street, New York 21, N.Y.

The first hearing aid with 2 crystal microphones

The Paravox "TOP-twin-tone"
***hears* through the top**

Tiny, with twin crystal microphones top-mounted to avoid surface noise and give balanced tone. This Paravox model has wide range and great sensitivity, permits a remarkable degree of fitting accuracy for varied hearing needs.

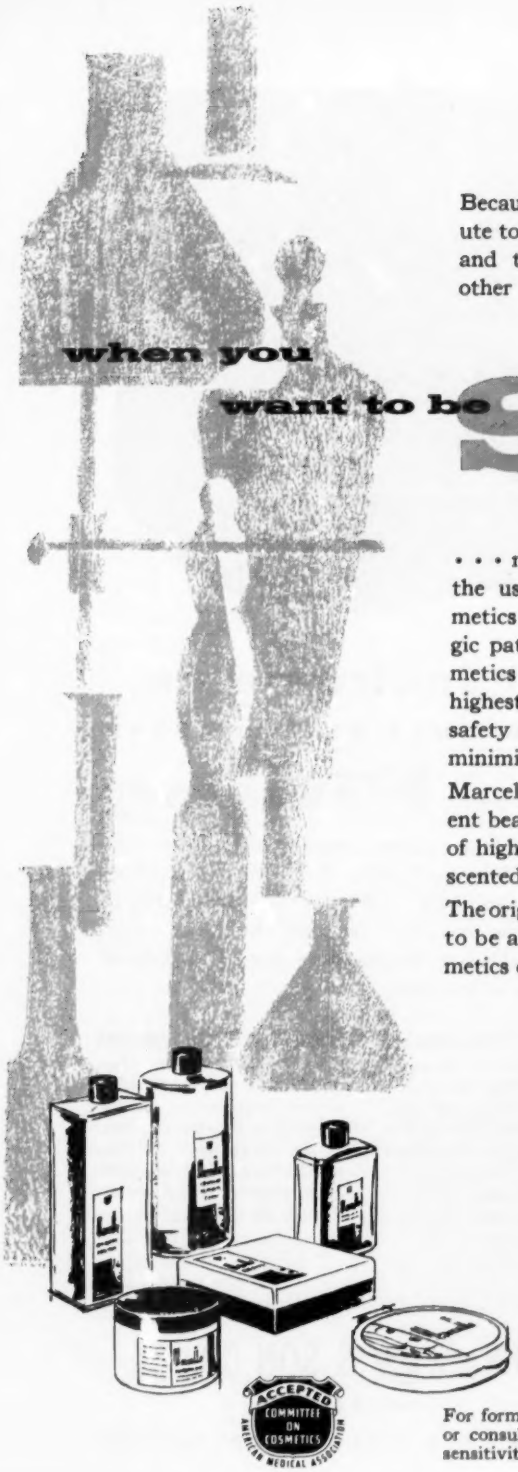
The "TOP-twin-tone" and other Paravox Hearing Aids * are exhibited at leading medical conventions annually, and are accepted by the Council on Physical Medicine and Rehabilitation, American Medical Association.

* Models D, J, Y-YM, YC, YC-7

WRITE FOR LITERATURE describing the "TOP-twin-tone" in greater detail.

PARAVOX FOR SOUND QUALITY

PARAVOX, INC.
2056 East Fourth St. **Cleveland 15, Ohio**



Because cosmetics may provoke or contribute to edema of the nasal mucous membrane and turbinates, inhalation difficulties and other respiratory allergic reactions • • •

when you want to be SURE

• • • many physicians routinely recommend the use of Marcelle Hypo-Allergenic Cosmetics to *protect* their treatment of the allergic patient, Marcelle Hypo-Allergenic Cosmetics are compounded according to the highest standards of purity, quality and safety with known allergens and irritants minimized or eliminated.

Marcelle's entire line of more than 40 different beauty preparations in a complete range of high fashion shades is available in either scented or unscented form.

The original Hypo-Allergenic Cosmetics. First to be accepted by the Committee on Cosmetics of the American Medical Association.

Marcelle
HYPO-ALLERGENIC
COSMETICS

FOR SENSITIVE AND ALLERGIC SKINS

ACCEPTED COMMITTEE ON COSMETICS AMERICAN MEDICAL ASSOCIATION

For formulae of Marcelle products, testing materials or consultation concerning special cases of cosmetic sensitivity, write to:

MARCELLE COSMETICS, INC., 1741 NORTH WESTERN AVENUE, CHICAGO 47, ILLINOIS

Authentic Holinger Instruments

FOR INFANTS AND CHILDREN

made only by **Pilling**

These new instruments are examples of the well-known Pilling excellence. Such "feel" could be achieved only by master craftsmen. Always made to the designer's original and exact specifications, Pilling instruments are often copied but never duplicated.

HOLINGER ANTERIOR COMMISSURE LARYNGOSCOPE

Permits better vision, easier exposure, more working room —less trauma. With two extra small light carriers and two extra small lamps.

Br 205 Special Infant Size
Br 207 Special Child Size

HOLINGER INFANT BRONCHOSCOPE

Reduced external diameter lessens possibility of trauma. Extra small lamps and light carriers without loss of illumination. Oxygen connection.

Br 900	3 mm x 20 cm
Br 905	3½ mm x 25 cm
Br 910	3½ mm x 30 cm
Br 915	4 mm x 30 cm
Br 920	5 mm x 30 cm

HOLINGER INFANT ESOPHAGOSCOPE

Special esophagoscope for foreign bodies—discs and other objects lodged in the cervical esophagus. Reaches the level of the foreign body without disadvantage of excess length. Special forceps also available.

Br 397 6 mm x 20 cm

THE AUTHENTIC INSTRUMENT IS AVAILABLE ONLY FROM PILLING—ORDER DIRECT

GEORGE P. **Pilling** & SON CO.

3451 WALNUT STREET PHILADELPHIA

HEADQUARTERS FOR INSTRUMENTS FOR BRONCHOESOPHAGOLOGY

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 59 JUNE 1954 NUMBER 6

COPYRIGHT, 1954, BY THE AMERICAN MEDICAL ASSOCIATION

A NEW TREATMENT FOR CHRONIC SECRETORY OTITIS MEDIA

B. W. ARMSTRONG, M.D.
CHARLOTTE, N. C.

THIS PRELIMINARY report illustrates the successful application of a very simple and inexpensive procedure in the treatment of chronic secretory otitis media. A small piece of plastic tubing,* measuring about 1.5 mm. in diameter, is inserted into the middle ear through a conventional myringotomy incision. This provides continuous ventilation of the middle ear and allows the lining membrane to return to normal. It also permits drainage of fluid into the external auditory canal and prevents the incision from closing prematurely. The tube may be left in place as long as desired—two to three weeks has been long enough in most cases. The tubing is inert, and no evidence of irritation or unfavorable reaction has been observed.

TECHNIQUE

A conventional myringotomy is performed and followed by repeated suction and inflation to remove as much fluid as possible. A piece of tubing about 1 cm. long is cut with a 45-degree bevel on one end. The beveled end is inserted through the incision in the tympanic membrane in such a way that the lip of the bevel is posterior (Fig. 1). The patient is instructed to practice Valsalva inflation several times each day and return in one week.

Inflation will determine patency of the tube and if there is any fluid present. The sound heard during inflation is of much lower pitch than that usually heard with a perforation in the tympanic membrane. Smaller tubing was used at first, but it was discarded because it sometimes became plugged with dried secretions. This has not happened since the change to the use of larger tubing, but if such plugging should occur, it is a simple matter to replace it with a new piece.

The tubing is left in from one to four weeks, depending on the duration of the disease and the clinical response. The perforation closes in about one week after the tubing is removed.

Five patients have been successfully treated by this method. A typical case is reviewed to illustrate the treatment.

REPORT OF A CASE

Mr. C. B. D., a 59-year-old industrialist, was seen in consultation because of persistent secretory otitis media involving his right ear. For six months he had recived regular treatment along the lines that are generally accepted. The ear had been inflated repeatedly, and a myringotomy had been performed on numerous occasions. Nose drops and antihistamines had been employed, without apparent benefit. Cortisone had been administered, without any improvement. There had been no evidence of allergy or sinus disease.

Examination revealed a right ear that was typical of secretory otitis media, with mixed deafness of 31.8% (conductive loss with a perceptive loss above 2000 cps). The left ear was normal in appearance and had a perceptive loss above 2000 cps of 13.9%. The rest of his examination was well within normal limits. Roentgenograms of the paranasal sinuses were normal.

From the Charlotte Eye, Ear and Throat Hospital.

* No. 444 T B-D plastic tubing (special vinyl compound) is used. This may be sterilized by boiling or autoclaving. Distribution by Becton, Dickinson, & Company, Rutherford, N. J.

A myringotomy was done and a moderate amount of thin, straw-colored fluid was removed by suction and inflation. A smear of the fluid showed an abundance of epithelial cells, but no other cellular elements and no bacteria were found. The ear "closed up" after two days, and one week later myringotomy was repeated. After the fluid had been removed, a piece of vinyl tubing was inserted through the incision, as described above. The ear remained comfortable, and improvement in hearing was maintained. Fifteen days later, with the tube still in the middle ear, an audiogram revealed a perceptive loss of only 13.9%. The tube was removed, and three days later the perforation had closed. After three weeks, his symptoms returned after an acute upper respiratory infection. Again myringotomy was done, a small amount of fluid was removed, and a tube was placed in the ear as before. This time the tube was left in for a month. During that period he had no symptoms referable to the ear, and there was no return of fluid. He has remained symptom-free since that time.

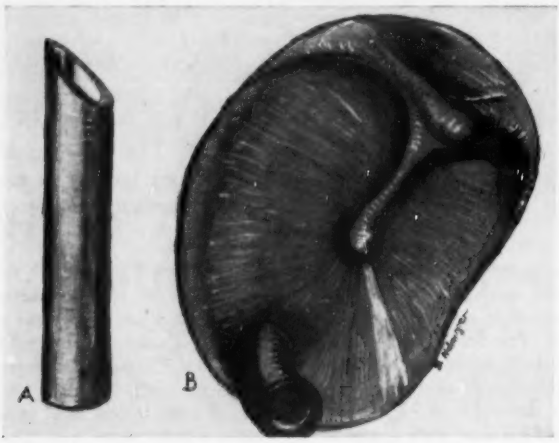


Fig. 1.—*A*, piece of plastic tubing showing the bevel; *B*, tympanic membrane with tube in place.

COMMENT

This method is not presented as a panacea in the management of secretory otitis media. It has been used only in chronic cases that have resisted treatment. The nasopharynx, nose, and sinuses must be treated when indicated. If the disease continues in spite of treatment and elimination of contributing conditions, this method, which provides continuous ventilation of the middle ear, may be considered. It may be assumed that a permanent perforation in the tympanic membrane can be obtained if the tube is left in over a long period of time.

SUMMARY

A new treatment for chronic secretory otitis media has been presented and its application demonstrated in a typical case. The treatment consists of placing a small plastic tube in the middle ear. This provides continuous ventilation of the middle ear, permits fluid to escape, and allows the lining membrane to return to normal. In most cases the tube is removed in two or three weeks, and the incision usually closes within a week after the tube is removed. No unfavorable reactions or complications have occurred.

TREATMENT OF NASAL POLYPI, WITH COMMENTS ON FRONTAL SINUS SURGERY

J. A. HARPMAN, M.S., B.Sc., M.B. (London), F.R.C.S. (Edinb.), D.L.O. (Eng.)
WARWICKSHIRE, ENGLAND

IN THIS paper there are discussed and described methods for curing nasal polyposis. Not all nasal polypi, of course, are of the same nature. Neither antrochoanal polyp nor carcinoma of the nose and sinuses presenting as polypi is discussed. The usual types of nasal polypi are of either allergic or infective origin. I have several times found allergic nasal polypi, if small and early in their development, to disappear under local or general antiallergic treatment; such disappearance, however, is unusual, and, as a rule, surgery is required to effect a cure. It is often not possible to draw a hard and fast dividing line between allergic and infective polypi, as infection often becomes superimposed on nasal sinus allergy.

LESS RADICAL SURGERY FOR NASAL POLYPOSIS

Cure of nasal polypi is dependent on removing or otherwise treating the diseased tissues from which the polypi arise. The extent of the surgery required for this depends upon the severity of the condition; thus, when only a few nasal polypi are present, cure may follow their simple removal; recurrence of the polypi is less likely if their removal is followed by gentle curettage of their base. In many cases, especially of elderly patients, a satisfactory procedure is removal of the polypi, intranasal ethmoidectomy, and sometimes removal of most of the middle turbinate, depending on the extent of its involvement by the disease. In such a case, a suitable method of intranasal ethmoidectomy is to open the ethmoid cells with upcutting nibbling forceps and to complete the operation by curettage, as illustrated by Portmann (1939). A curette for this procedure is illustrated in Figure 1. This method of intranasal ethmoidectomy I have found to be better than others. It has been my experience that ethmoidectomy is curative in a number of cases of nasal polyposis but unsatisfactory when there is also much suppuration; it may be dangerous when there are bony dehiscences in the roof or lateral walls of the ethmoid labyrinth—I have encountered one such case. Intranasal ethmoidectomy can but very rarely be complete. When there is purulent ethmoiditis, suppuration continues after incomplete ethmoidectomy, and then especially nasal crusting may occur. Intranasal ethmoidectomy may be followed by cicatricial obstruction of the frontonasal duct.

Occasionally, cases of nasal polyposis are encountered in which the polypi arise from the antra, the other sinuses being normal. The Caldwell-Luc operation with removal of the antral mucosa is generally curative in such cases, but I have had two patients in whom the antra refilled twice with polypi after this surgery but in whom the condition was finally arrested by deep x-ray therapy to the antra after again

Consultant Ear, Nose and Throat Surgeon, Warwick, Stratford-on-Avon, and Shipston-on-Stour Hospitals.

removing the diseased lining. This method of radiotherapy is safer than use of radium needles, as, owing to the irregular size and shape of the cavity to be treated, it is difficult to avoid, with these, irradiating some areas too much and some too little. Excessive irradiation may result in very painful necrosis.

I have tried "transantral" ethmoidectomy in many cases. The transantral approach to the ethmoids is really adequate only for the lower part of the middle and posterior ethmoid cells; it is not possible to approach anterior ethmoid cells through the antrum without risking damage to the lachrymal duct. "Transantral ethmoidectomy" is really a combination of transantral and intranasal ethmoidectomy.

Nasal polyposis is cured occasionally by intranasal ethmoidectomy and partial removal of diseased antral lining through an intranasal antrostomy.

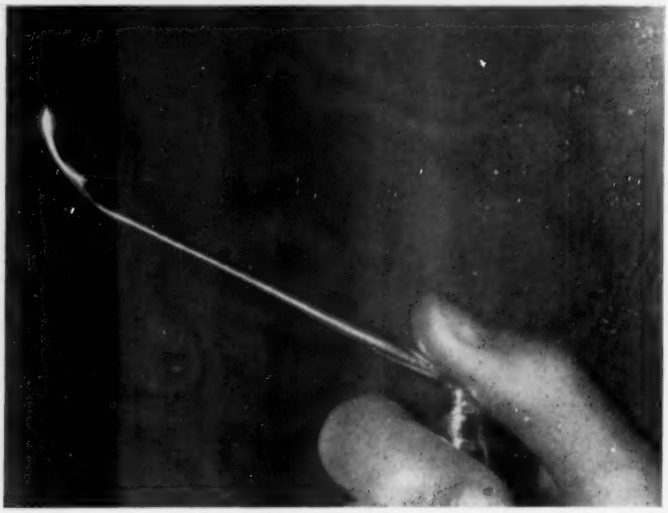


Fig. 1.—Ethmoid curette.

RADICAL SINUS SURGERY FOR GROSS CHRONIC POLYPOID PURULENT POLYSINUSITIS

There are a number of patients all of whose nasal sinuses are full of polypi and often also pus; often the best treatment for these is removal of the diseased lining from all the affected sinuses. There is much difference of opinion as to whether or not sinus mucosa should be removed. My experience leads me to suggest that when a nasal sinus is sufficiently diseased to warrant radical surgery its lining is so diseased that the operation will not be fully successful in curing the patient's complaints unless this lining is removed. In general, it seems to me that when a nasal sinus is opened by a radical approach and the lining in it is found not sufficiently diseased to require removal that approach should not have been used.

The radical operations I have found to give the best results in chronic polypoid purulent polysinusitis not cured by simpler measures have been two combinations: the Ferris Smith (1933, 1935), Lynch, Sewall, or Luongo (1936) (Neivert, 1942) type of operation and the Caldwell-Luc operation, and the Ferris Smith and Norman Patterson (1939, 1942, 1944, 1945) types of operations. At present I

favor the second combination because it permits more thorough exenteration of the inferior ethmoids and because the same approach can be used for the antrum as well as for the other sinuses. The Patterson operation alone, I think, has the disadvantages of not permitting adequate access to the higher ethmoid cells and of not dealing adequately with the frontal sinus and frontonasal duct. An operation resembling the Ferris Smith and part of the Patterson operation was described by Jacques and Durand (1903).

Chemotherapy.—I usually perform these operations under cover of penicillin and sulfonamides or chlortetracycline (Aureomycin) systemically given before, during and for a few days after operation.

Anesthesia.—In a few patients with very troublesome chronic bronchitis or bronchiectasis, I have performed the operation with local anesthesia, using cocaine in the nose in the usual way and infiltrating the operation area with procaine and epinephrine solution. The procedure under local anesthesia is, however, quite an ordeal for most patients, and for some years now at Warwick Hospital we have had no case in which it has not been possible to perform it comfortably with use of general anesthesia. Patients suffering also from bronchiectasis are treated by postural drainage preoperatively and if necessary their trachea and bronchi are sucked at operation.

The eyebrow is generally clipped or shaved; it is often advisable to keep the eye closed with a suture during the operation.

The Incision.—Figure 2 and 3 are photographs of patients, showing the incision shortly after operation. The incision is placed far forward so that it lies over bone that is left; if the incision is placed more posteriorly so that it comes to lie over areas where bone has been removed, it may be pulled in and the eventual cosmetic result may not be so satisfactory; for this reason I have abandoned Patterson's original incision. The extent of the upper part of the incision depends upon the size of the frontal sinus. If this is large, I extend the incision through the eyebrow up to the supraorbital notch; by not extending it farther, the supraorbital vessels and nerve are preserved. The lining from lateral extensions of a large frontal sinus can in most cases be removed by working "around the corner" without additional exposure, but occasionally in the case of a very large frontal sinus wider exposure may be required. The incision lies in front of the angular vessels. Some blood vessels that run across the upper part of the nasal bridge (Figs. 5, 6, 7, 9, 10, 16, 17, 18) have to be divided and ligatured. If the incision is made down to bone very gradually, it is sometimes possible to pick up these vessels before dividing them. Some smaller bleeding points in the incision also often require controlling. The incision is hemorrhagic, and its adequate performance is aided by use of suction. In the lower part of the incision it is often possible to go down to bone between muscle fibers rather than through them.

Periosteal Elevation.—In most cases Freer's nasal septum elevator is safer than elevators with square ends and sharp points. The orbital periosteum is separated from the medial wall of the orbit backward to the anterior ethmoidal vessels and nerve and from the floor of the frontal sinus—not usually laterally to the supraorbital notch—downward to the floor of the orbit. The medial palpebral ligament should first be divided with a knife as far medially as possible, cutting against the bone (Figs. 4 and 5). The whole tear sac should be lifted outward from its bony bed (Fig. 6). This is nearly always quite easy provided the elevator, sometimes aided by

pledgets of gauze, is employed here from above downward and slightly forward; otherwise, the sac might be injured. This elevation is continued downward to the beginning of the tear duct. Either with the same elevator or with an elevator such as Lempert's broad mastoid periosteal elevator a small area of the maxillary bone just below the medial part of the inferior orbital margin is denuded (Fig. 7). This subperiosteal elevation should not be carried too far laterally, so as not to injure the infraorbital nerve and vessels. The next step is to elevate the periosteum from the anterior half or so of the medial part of the orbital floor with a Freer elevator. This requires freeing the origin of the inferior oblique muscle, and this step requires

Fig. 2.—Patient with large frontal sinuses, showing the skin scar of a right Ferris Smith and Patterson type of operation performed some months previously.

Fig. 3.—Patient with small frontal sinuses, showing the cutaneous scar of a left Ferris Smith and Patterson type of operation performed 10 days previously.

patience and care (Fig. 8). The muscle also arises from the lateral part of the junction of the tear sac with the tear duct. Detachment of the inferior oblique muscle is often helped much by careful use of a fine scalpel, e. g., the No. 15 Bard-Parker blade (Fig. 8). A small blood vessel perforating the upper part of the lachrymal bone and another one perforating the superomedial part of the anterior wall of the maxilla sometimes bleed but soon stop bleeding on application of pledgets of gauze or wool soaked in epinephrine or thrombin solution to them.

Retraction and Commencing the Bone Work.—I do not find self-retaining retractors very satisfactory in this operation and prefer to use a malleable dural elevator and a Woodman palate retractor (Figs. 4, 10, 17, and 18).

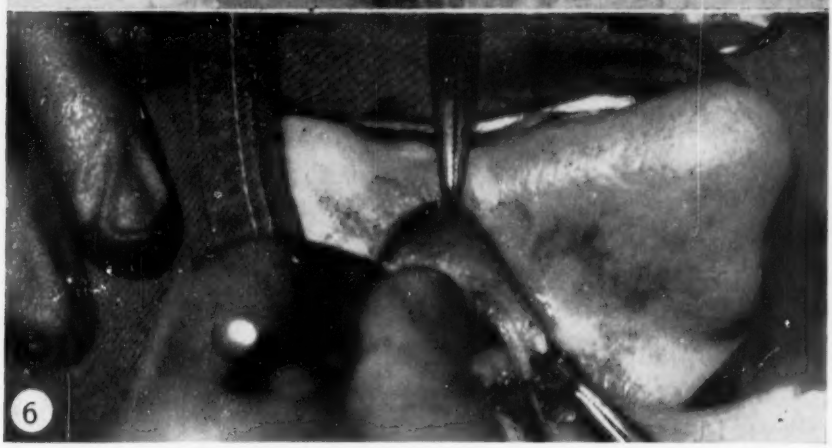


Fig. 4.—Right medial palpebral ligament, just above a sucker end.

Fig. 5.—Right medial palpebral ligament being divided; a sucker end is being held close to the scalpel to maintain the field bloodless.

Fig. 6.—Right tear sac being displaced outward from its bony bed with a Freer elevator.

The lachrymal bone is perforated with a pointed instrument; occasionally it is fairly thick, and then it is best perforated with a mallet and gouge. Through a small opening thus made a small rongeur (Kerrison's is especially suitable) is introduced (Figs. 9 and 10) and most of the lachrymal bone, the frontonasal process of the maxilla, the descending process of the frontal bone, the floor of the frontal sinus up

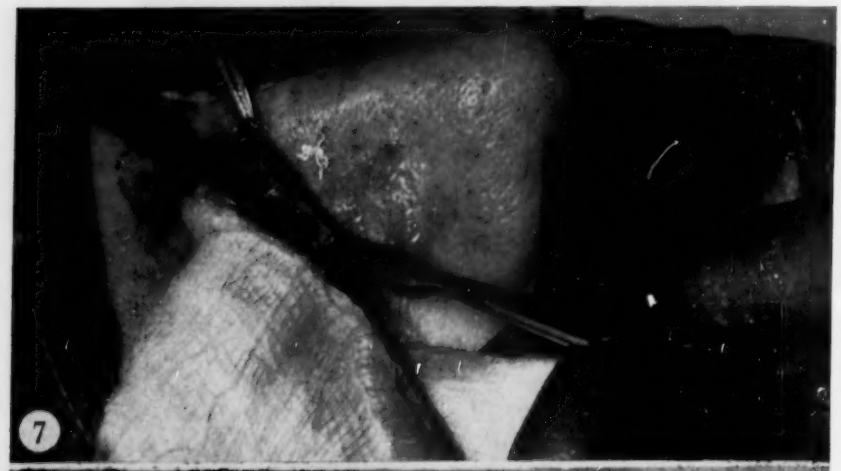


Fig. 7.—Anterosuperior medial part of the right superior maxilla being denuded with a broad Lempert periosteal elevator. The instrument seen in the upper part of the photograph is a sucker end.

Fig. 8.—Right inferior oblique muscle being freed at its origin with a small periosteal elevator and a scalpel. The tear duct is the clearly seen light structure.

to the supraorbital notch, the anterior part of the lamina papyracea, and very occasionally the most lateral fringe of the nasal bone are removed (Figs. 11, 16, and 18). The extent of lamina papyracea to be removed depends upon the case; it is not usually necessary to remove it farther back than the anterior ethmoidal foramen, but

Fig. 9.—Photograph showing how in a left-sided operation the bone removal is begun with a Kerrison bone forceps at the upper part of the lachrymal bone.

Fig. 10.—Photograph showing how, in a right-sided operation, the bone removal is continued with a Kerrison bone forceps, the operation field being kept clear by suction. The photograph also shows how the medial orbital periosteum is held laterally with a malleable dural type of elevator.

in some cases access to posterior ethmoidal cells is inadequate unless the lamina is removed backward nearly 1 cm. beyond the anterior ethmoidal foramen. Occasionally it is advisable to place a ligature around the anterior ethmoidal blood vessels; they can be controlled more easily with a Cushing silver clip, which I have used often without evil results from leaving it in place (Figs. 12 and 13). It is only very rarely necessary to divide these vessels to obtain satisfactory access to the hinder reaches of the ethmoid labyrinth. Ferris Smith (1933, 1935) described his method of ligating the posterior ethmoidal vessels in this operation and stated that the anterior ethmoidal vessels could be divided and ignored, as they soon stopped bleeding spon-

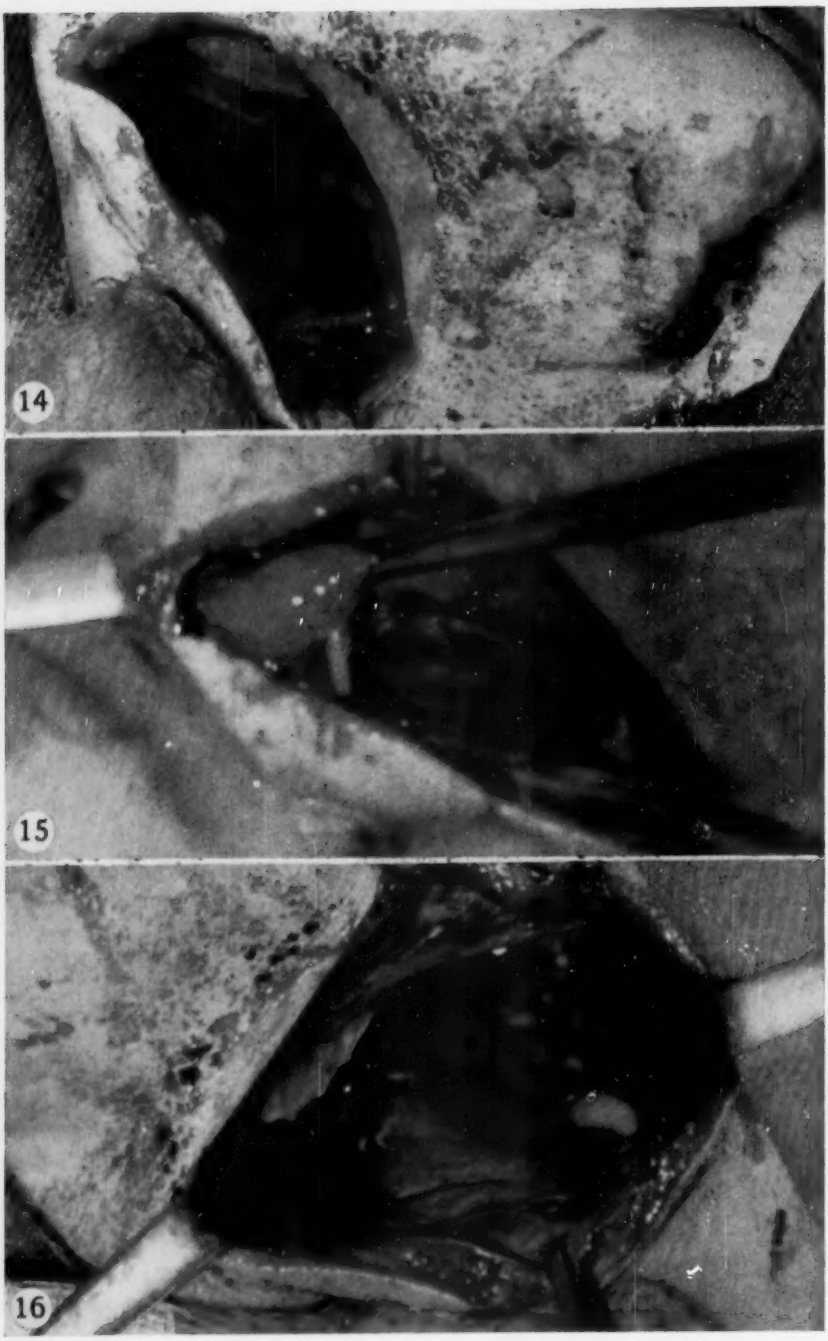
Fig. 11.—Skull on which there have been outlined the smaller or greater areas of bone removed in various cases of combined left Ferris Smith and Patterson operations.

taneously after division. This mistake is being quoted in several recent textbooks. In the pictures illustrating Ferris Smith's paper it is obvious that the vessels being ligated are actually the anterior ethmoidal ones; in this operation the orbital periosteum is but very rarely elevated as far back as the posterior ethmoidal foramen, and it should be borne in mind that the optic nerve enters the orbit only about 1 cm. behind this foramen.

Clearing out the Frontal Sinus.—It is only very rarely necessary to remove the floor of the frontal sinus lateral to the supraorbital notch to be able to remove its lining thoroughly. It may be necessary to remove the floor of the sinus right up to the supraorbital ridge for access purposes. It is important to remove most of the shelf of frontal bone that extends backward in the anteromedial inferior part of the

Fig. 12.—Anteroposterior skull roentgenogram showing a Cushing silver clip on the anterior ethmoid vessels at the anterior ethmoid foramen.

Fig. 13.—Lateral skull roentgenogram showing a Cushing silver clip on the anterior ethmoid vessels at the anterior ethmoid foramen.



Figures 14, 15 and 16

(See legends on opposite page)

frontal sinus (Fig. 14); otherwise, "pocketing" may occur above and medial to this projection. This removal should be done with care, owing to the proximity of the cribriform plate. The mucosa of the frontal sinus is then completely removed (Figs. 15 and 16) by wiping with small pledgets of gauze. Lateral and upward extensions of the sinus can be cleared by holding these pledgets on suitably curved forceps.

Exenteration of the Ethmoid Labyrinth.—The ethmoid cells are broken into and the ethmoid labyrinth turned into one cavity from which all mucosa is removed. This is suitably carried out with the small and large Kerrison bone forceps and pledgets of gauze. When this has been done adequately, the floor of the anterior fossa of the skull forming the roof of the ethmoid labyrinth should be evident as bare bone and often the anterior ethmoidal blood vessels and nerve are seen coursing across its anterior part. The remaining part of the lamina should appear denuded of mucosa. Care is required in the posterosuperior part of the ethmoid labyrinth, owing to its proximity to the optic nerve. The lateral part of the middle turbinate is part of the ethmoid labyrinth, and in nearly all cases in which radical ethmoidectomy is required the procedure cannot be accomplished adequately without removing most of the middle turbinate with turbinotomy scissors; sometimes it is helpful to remove the most posterior part of the middle turbinate with Kerrison's bone forceps. The uppermost part of the middle turbinate is preserved (Fig. 16), but the mucosa from its lateral aspect is removed. Just in front of the middle turbinate it is sometimes necessary to remove, or to incise, some of the lateral nasal mucous membrane.

Opening and Clearing Out the Sphenoid Sinus.—After removal of the posterior part of the middle turbinal, the sphenoid ostium is usually visible. Probing the sphenoid sinus through the ostium often helps to decide whether the sinus does or does not contain pus and thickened lining. Should these be found, the whole anterior wall of the sphenoid sinus is removed with punch forceps, such as those of Kerrison or Ferris Smith. I have not found it necessary to ligate and divide the sphenopalatine artery and to remove also the sphenoid sinus floor, as recommended by Ferris Smith; to do so produces an unnecessarily large cavity. The sphenoid sinus lining can now be readily removed with forceps and by wiping it out with pledgets of gauze; the good exposure in the operation renders this quite safe; in one of my cases the bony lateral sphenoid wall was absent, the cavernous sinus lining being visible; the cavernous sinus was not injured.

Opening the Maxillary Antrum (Figs. 16, 14, 11, 10).—Patterson described how the antrum could be opened and the bone removed from around the lachrymal duct by chiseling the medial part of the intraorbital margin. This method I have found somewhat dangerous, and I prefer to remove the bone from around the upper

---

EXPLANATION OF FIGURES 14, 15 and 16

Fig. 14.—Shown in the lower part of the operation field are polypi in the right antrum bulging outward just below the tear duct, and also the bony shelf just below the anteromedial part of the frontal sinus that will have to be partly removed to prevent later "pocketing" in this sinus.

Fig. 15.—A polyp being pulled out of the right frontal sinus.

Fig. 16.—Photograph taken towards the end of a left-sided operation. It shows the cavities of the cleared-out frontal, ethmoid, and maxillary sinuses; the upper remaining part of the middle turbinate from the lateral aspect of which diseased mucosa has been removed; the tear duct; the tear sac and, above and behind the latter, some orbital fat protruding through a small tear in the orbital periosteum.

part of the tear duct, of the superomedial part of the anterior wall of the antrum and of the anteromedial part of its roof, and the antronasal wall above the inferior turbinate by patient, careful, and slow working with various bone rongeurs; the bone around the upper part of the tear duct is most safely removed with a small Kerrison bone forceps. I have on no occasion damaged the tear duct by this method.

Fig. 17.—Photograph taken during removal of the right antral lining with Watson-William's polyp forceps. These are introduced into the antrum either below and lateral, or, as when this photograph was taken, above and medial to the tear duct.

Fig. 18.—Photograph taken toward the end of a right-sided operation before packing the operation cavity and suturing the wound. The tear sac and duct and some of the extent of bone removal are seen.

Clearing Out the Antrum.—All the diseased lining from the antrum is then completely removed with instruments such as the polyp forceps of Watson-Williams (Fig. 17) and by wiping with pledgets of gauze on curved forceps; after a little practice this can be accomplished as readily as by the Caldwell-Luc approach. The

middle meatal antronasal wall is then removed. Some inferior and posterior ethmoidal cells are cleared out more readily at this stage. There is no need to make an antronasal window in the inferior meatus.

Polyposis of the Nasal Septum.—Occasionally the mucosa of the nasal septum, usually superiorly, is polypoid (also Patterson, 1945; others). The affected mucosa should then be removed.

Dressing the Operation Cavity.—When there is much bleeding I usually pack the operation area loosely with ribbon gauze impregnated with bismuth, iodoform, and paraffin paste, which is gradually removed through the nose on the days after operation. If there is not much bleeding I apply pieces of self-absorbent gelatin sponge to oozing areas and leave them. The gelatin sponge may be moistened before application and this is advantageously done with thrombin solution, although even if only isotonic sodium chloride solution is used the gelatin is still effectively hemostatic provided the bleeding is only slight.

Suture of the Wound.—To secure sound healing of the wound without fistula formation, it is advisable to suture the divided periosteum and muscle as well as the skin. I generally include all the layers in fine interrupted nylon sutures. Alternatively, the periosteum and muscle can be sutured with absorbable surgical suture in one layer and the skin separately with nylon. I have given up this practice, however, as I have found that the more absorbable surgical suture is buried in the wound the greater are the chances of wound suppuration. The skin sutures are left no longer than 48 hours and should not be tied too tightly, so as not to leave stitch marks.

Dressing the Wound.—The eye is irrigated and the wound and eyelids covered with gauze impregnated with petrolatum. Pledgets of wool are applied over this, then another piece of gauze, and then a firm bandage; a firm bandage is kept on for about three days and helps to reduce the amount of eyelid edema that may develop postoperatively.

Postoperative Nasal Treatment.—The patient is advised not to blow her or his nose for some ten postoperative days. Should there be any blood crusts in the nostril, some ointment is put into it. Many patients develop some crusty discharge in the nasal cavity during the first few months following the operation; olive oil nasal drops help this condition and the crusts can be picked out from time to time with forceps. In all my cases except one this crusting ceased after a few months, when the operation area had become epithelized. In one case it continued until the nasal cavity on the operated on side had been made narrower by submucous resection of the nasal septum; there had been gross septal deflection to the opposite side. The operated on sinuses are eventually partly filled with new bone and fibrous tissue, so that by the time they are epithelized their cavities and their openings into the nose have become much smaller.

SOME OBSERVATIONS ON THE SURGERY OF THE FRONTAL SINUS

There has been much controversy on the advisability of removing the frontal sinus lining in external operations on this sinus. My experience hitherto leads me to conclude that when on opening a frontal sinus externally the mucosa in it is found to be so little diseased that it can be expected, if left in place, not to continue suppurating and producing polypi, an error of judgment has been made and that

sinus should not have been opened externally. On the other hand, leaving a lining that is so diseased that it will continue suppurating and producing polypi may lead to disaster. Thus, I have seen the newly made frontonasal opening after such an operation become blocked by a newly developed polyp from the frontal sinus; this led to retention of pus in the sinus and to eventual death of the patient from intracranial suppuration.

I have found that when the mucosa has been completely removed from a small frontal sinus, the whole cavity of the frontal sinus becomes filled with new fibrous and bony tissue as healing takes place (also Lieberman and Babb, 1948); when the frontal sinus is large healing results in there remaining only a small cavity. In a number of cases, especially if the frontal sinus is small, healing after removal of the lining results in there being no more patent frontonasal duct, for which there is no more need when the whole frontal sinus has become filled with new fibrous and bony tissue. The situation is then somewhat similar to that which occurs in the mastoid and aditus following cortical mastoidectomy. I have found that filling a cortical mastoidectomy cavity with a paste of plasma powder and penicillin solution (Harpman, 1950) leads to the operation cavity becoming filled with firm fibrous tissue which appears to resist reinfection. I am now filling large operated-on frontal sinuses with such a paste and trying to ascertain whether this is beneficial to firm and rapid healing.

The course of healing after the Lothrop (Neivert, 1942) type of operation is of interest in deciding on the management of infected frontal sinuses; the following three case histories are illustrative:

An elderly man had a fistula in the center of his forehead, leading into both heavily infected frontal sinuses. He was suffering from headaches and much pus was exuding from the fistula, around which there was low grade osteomyelitis. His other nasal sinuses were surprisingly free from infection. I excised the fistula, removed the bony anterior walls of his frontal sinuses and removed their grossly diseased lining; I then removed the frontal intersinus septum and the upper anterior part of the nasal septum as recommended for the Lothrop operation, thus making a large opening from both frontal sinuses into the nose. The opening was enlarged further by removing upper anterior ethmoid cells from above; the incision was then sutured. The patient was given penicillin systemically. The fistula healed. A few months later it was found that the frontal sinus cavities had become completely filled with fibrous tissue and that this encroached upon and had closed the opening—for which there was now no more need—from the frontal sinuses into the nose.

A patient had a subacute pansinusitis, worse in both frontal sinuses, osteomyelitis of the frontal bones, and an extradural frontal abscess. I removed the anterior and posterior walls of both frontal sinuses, removed the frontal sinus linings down to the frontonasal ducts, filled the operation cavity with a paste of penicillin solution and plasma powder, sutured the wound, and gave him penicillin and sulfonamides systemically. His antra were washed out. The infection subsided completely and later gentle probing of the frontonasal ducts revealed complete obliteration of their upper parts; the forehead was firmly healed.

A patient had a large osteoma in a frontal sinus; it had blocked the upper part of the frontonasal duct and the small parts of frontal sinus cavity that were not full of osteoma were full of pus and infected mucosa. I had to remove the anterior wall of the frontal sinus to remove the large osteoma; the posterior wall and the floor of the sinus were left intact; the mucosa was removed and the frontonasal duct was enlarged from above into the nose. The large operation cavity was lightly packed with ribbon gauze that was led through the enlarged frontonasal duct into the nose, through which it was removed gradually during the days following the operation. Some months later it was found that the operated-on frontal sinus and its opening into the nose had been completely filled by scar tissue.

Experimental confirmation of these findings has been obtained by Walsh (1943).

I have seen a number of patients whose infected frontal sinuses have been operated on by the Harmer-Russell (1931) procedure. In some of these the enlarged frontonasal duct was blocked intermittently by polypoid frontal sinus mucosa and these patients were only cured of their symptoms of frontal sinus suppuration by removing all this mucosa. In my experience, after transnasal manipulations in the frontonasal duct, this is often obliterated by scar tissue. Medical literature abounds with descriptions of methods for maintaining the patency of a newly created opening from the frontal sinus into the nose. The first requisite is to remove sufficient bone at the correct site. Many cover the orbital periosteum that is left exposed in the nose with pedicled flaps of nasal mucosa (e. g., McNaught, 1936; Sewall, 1935; Bárány, 1924; Sourdille, 1930; Soderberg, 1938; others); this method I have tried and given up on finding pus accumulating under the pedicle (e. g., also Mygind,* 1938). In a number of cases I have applied a free graft of mucosa peeled off a piece of turbinal to the exposed orbital periosteum; no advantage appears to have accrued from this procedure.

I have reached the conclusion that when a frontal sinus is so infected that conservative measures fail to relieve it and it comes to require external surgery, the infected lining should be removed. Drainage of the sinus cavity into the nose after the operation is required only until healing has taken place. The operation described in this paper fulfills these criteria. Sebileau (1905) stressed that when all the diseased lining has been removed from a frontal sinus drains are not required.

COMPLICATIONS AND RESULTS OF THE RADICAL SINUS OPERATION DESCRIBED

The complications and results of the radical sinus operation described above obtained in some 50 cases are described and discussed here.

The cutaneous scar, while often almost invisible some months after the operation, has remained conspicuous in some cases and in a very few become slightly keloid. Eyelid edema during the first few postoperative days is becoming less frequent as more experience of the operation is acquired.

No matter how carefully the operation has been performed, occasionally a tear has occurred in the orbital periosteum, usually where lamina papyracea and frontal bone join; this accident has seemed more common in patients who have had much previous intranasal ethmoid surgery. Orbital fat has prolapsed through the tear (Fig. 16). Orbital infection has not occurred, but temporary diplopia resulted in four cases, the extraocular muscle most affected being, surprisingly, the external rectus. In two cases in which the amount of fat prolapsing was considerable, slight enophthalmos has resulted.

Some of my first few cases of Patterson's operation suffered from postoperative epiphora lasting at most a few weeks; this complication does not occur any longer in my cases. In one patient the tear duct was torn across by an assistant's misuse of a retractor; I then removed the medial wall of the tear sac and the dacryocystorhinostomy thus performed prevented the patient's having any symptoms from the accident.

* Mygind, S. H.: Cited by Soderberg.

Some patients suffered from temporary postoperative neuralgias and paresthesiae about the face and head; in only one case did these symptoms last longer than a few months; they were usually mild and relieved by mild analgesics.

During the first few months after removing diseased lining from a frontal sinus three of my patients, following either a cold or swimming, developed some reinfection in the healing cavity, producing swelling of the upper eyelid. Systemic sulfonamide and/or penicillin administration and use of an ephedrine solution nasal spray resulted in prompt subsiding of the infection. In one case this complication occurred about one year postoperatively, and the incision had to be partly reopened to let out some pus. It is probable that in these cases vestiges of diseased frontal sinus mucosa had been left behind at the original operation.

Intranasal suppuration has been reduced in all the cases and cured in many. It has persisted in some, especially in those suffering also from bronchiectasis or chronic bronchitis. It has been diminished, but not cured, in some cases of chronic purulent pansinusitis without polyposis in which the operation was tried. In many of the patients there has been some crusting of the nasal secretions during the first few months after the operation; the management of this complication has been discussed above. In many cases the operation has cured sinusitis pains and headaches.

No patient submitted to the radical sinus operation described in this paper has had recurrence of nasal polypi, nor have intracranial complications or osteomyelitis occurred. Experience of other cases leads me to suggest that such spread of sepsis is more likely to follow operations on infected frontal and ethmoid sinuses in which infected sinus mucosa is left.

The occurrence of postoperative complications of radical sinus surgery renders it imperative that such surgery is not done unless simpler measures have failed or will definitely prove unsuccessful in the given case.

SUMMARY

Nasal polyposis and polypoid sinusitis can be cured. The treatment employed should be adapted to each individual patient, the simplest and safest procedure likely to cure being used and radical surgery being reserved for cases that have not been or will not be cured by simpler means. In this paper there are described and discussed methods by which such cure is obtainable.

BIBLIOGRAPHY

Bárány, R.: Äussere Stirnhöhlenoperation mit Herstellung einer breiten Kommunikation zwischen Stirnhöhle und Nase mittels Schleimhautplastik: Schonung der Schleimhautauskleidung der Stirnhöhle, Acta oto-laryng. **7**:41, 1924.

Harmer, D., and Russell, H. B.: The Treatment of Frontal Sinusitis by Intubation: An Analysis of 63 Cases, J. Laryng. & Otol. **46**:384 (June) 1931.

Harpman, J. A.: On the Treatment of Otogenic Brain Abscesses, J. Laryng. & Otol. **64**:319 (June) 1950.

Jaques P., and Durand, A.: Du procédé de choix pour la cure radicale de la sinusite fronto-ethmoidale chronique, Ann. d. mal. de l'oreille du larynx **29**:129 (Aug.) 1903.

Lieberman, G. E., and Babb, J. W.: Nonregeneration of the Mucous Membrane of the Frontal Sinus After Its Surgical Removal (in the Dog), Arch. Otolaryng. **47**:421 (April) 1948.

Luongo, R.: The External Frontoethmosphenoidal Operation, Laryngoscope **46**:1 (Jan.) 1936.

McNaught, R. C.: A Refinement of the External Fronto-Ethmosphenoid Operation: A New Nasofrontal Pedicle Flap, Arch. Otolaryng. **23**:544 (May) 1936.

Neivert, H.: Surgery of the Nose and Sinuses, in Nelson, New Loose-Leaf Surgery, New York, Thos. Nelson & Sons, 1941, Vol. 7, p. 1.

Patterson, N.: External Operations on the Frontal and Ethmoidal Sinuses, J. Laryng. & Otol. **54**:235 (May) 1939.

External Operations on the Nasal Accessory Sinuses, Arch. Otolaryng. **36**:311 (Sept.) 1942.

Chronic Sinusitis with Polypi, a Radical External Operation, Lancet **1**:558 (April 29) 1944.

External Operation on the Nasal Sinuses, J. Laryng. & Otol. **60**:449 (Nov.) 1945.

Portmann, G.: Treatise on the Surgical Technique of Otorhinolaryngology, translated by Piere Viole, Baltimore, William Wood & Company, 1939.

Sebileau, P.: Reflexions sur la cure chirurgicale de la sinusite frontale chronique, Ann. d. mal. de l'oreille du larynx **32**:1 (Jan.) 1905.

Sewall, E. C.: Operative Treatment of Nasal Sinus Disease, Ann. Otol. Rhin. & Laryng. **44**:307 (June) 1935.

Smith, F.: Chronic Sinus Disease: Its Present Status, J. A. M. A. **100**:402 (Feb. 11) 1933.

Management of Chronic Sinus Disease—Conservative or Radical? J. Laryng. & Otol. **50**:604 (Aug.) 1935.

Soderberg, F.: Beitrag zur Kenntnis der Bárány'schen Stirnhöhlenoperation, Acta oto-laryng. **26**:541, 1938.

Sourdille, M.: Techniques chirurgicales nouvelles pour le traitement des surdités de conduction, Ann. d. mal. de l'oreille du larynx **49**:10 (Jan.) 1930.

Walsh, T. E.: Experimental Surgery of the Frontal Sinus: The Role of the Ostium and Nasofrontal Duct in Postoperative Healing, Laryngoscope **53**:75 (Feb.) 1943.

EXOGENOUS FOREIGN BODIES OF THE EXTERNAL AUDITORY CANAL, NASAL CAVITY, AND ESOPHAGUS

Report of Two Cases of Coins in the Esophagus Occupying the Sagittal Plane

J. C. PEELE, M.D.
KINSTON, N. C.

THE DIAGNOSIS and management of foreign bodies in the ears, nose, throat, and air and food passages constitute an integral and fascinating part of otolaryngologic practice. The problems presented may be simple in some instances and complex in others. Much progress has been made in this type of work since the turn of the century. Many sound principles of diagnosis and treatment have been established, preoperative and postoperative care improved, new instruments devised, and techniques of mechanical extraction and x-ray examination perfected. Physicians are better trained in the diagnosis of foreign bodies, and the general public is more mindful of the potentially dire consequences incident to their neglect. As a result of this progress there has been an astounding decrease in morbidity and mortality, and the number of external operations required for removal of foreign bodies in these regions has reached a new and gratifying low level.

While much has been accomplished in the management of foreign bodies in the ears, nose, throat, and air and food passages, the fact remains that the treatment of each foreign body patient must be to a certain extent individualized. At times accepted techniques of extraction must be altered, standard instruments modified, or new instruments devised in order to carry the treatment of some special cases of foreign bodies in the air and food passages to a successful conclusion. Added problems are often superimposed by the many difficulties confronting the otolaryngologist doing foreign body work in a small hospital.

The present paper concerns all exogenous foreign bodies actually encountered by clinical examination, x-ray, or both in the external auditory canal, nasal cavity, and esophagus from Feb. 1, 1948, through July 31, 1953. These patients were seen in an office practice that is predominantly private in nature. In the community of Kinston, N. C. there are no free clinics as there are in large urban areas, and patients of clinic status are seen in the daily office routine along with private patients. The population of this community during the period in question was approximately 18,000 to 20,000. The clinic group with which I am associated ordinarily draws enough patients from adjoining counties to bring this total up to approximately 50,000 to 75,000.

This study was undertaken primarily to provide a reasonably accurate determination of the incidence of foreign bodies seen in office practice in a small community, to record certain foreign body cases in the regions under consideration

Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

presenting interesting or unique features in certain phases of management, and, especially, to present two rare and unusual cases of coins lodged in the esophagus in the sagittal plane in conjunction with the mechanical problems of their extraction.

INCIDENCE

Clinical impressions gained regarding the frequency of any given disease are frequently erroneous unless these impressions have been influenced by a consideration of accurate statistical data. The statement is often heard and sometimes read that foreign bodies of the ear, nose, and throat region are common; yet the authors of these verbal or written statements fail to provide supportive evidence. Publications in the literature calculated to establish a definite incidence of exogenous foreign bodies in the external auditory canal, nasal cavity, and esophagus are virtually nonexistent.

Bezold and Siebenmann [1] stated that one case of foreign body in the ear canal is seen to every 60 or 70 ear patients, excluding cases of cerumen. Griffin [2] found that out of 11,216 aural cases treated only 101 were treated for foreign bodies in the ear. Keen [3] examined the records of 15,000 children treated at the School Clinic of Leicester during the nine-year period prior to 1930. In this number of 15,000 children there were 43 unsuspected foreign bodies of the external auditory canal and 30 unsuspected nasal foreign bodies. Hung and Lin [4] stated that esophageal foreign bodies constituted only 0.28% of all cases in which treatment was given in the Department of Otolaryngology, National Taiwan University Hospital during 1946-1950. They included references regarding the incidence of esophageal foreign bodies from the literature.

During the period covered by this study a total of 6,983 new patients were seen in office practice. It is my custom to do a thorough examination of the ears, nose, and throat on all new patients unless occasional circumstances dictate a less complete examination. A review of these records reveal that a satisfactory examination of the aural canals was recorded in 6,805 new patients and that a satisfactory examination of the nasal cavity was made on 6,803 new patients. There were 48 patients with 52 foreign bodies in the external auditory canal; 17 patients with 18 nasal foreign bodies; 38 patients with foreign bodies of the esophagus. Thus the incidence for foreign bodies in the external auditory canal was found to be 0.007%; for nasal foreign bodies, 0.0025%; for esophageal foreign bodies, 0.0054%.

EXOGENOUS FOREIGN BODIES IN THE EXTERNAL AUDITORY CANAL

Most foreign bodies in the external auditory canal are seen promptly, removed easily, and occasion few symptoms or pathological changes. They may be animate or inanimate; unilateral or bilateral; single or multiple. Statistical studies of both animate and inanimate foreign objects removed from this region bear a striking similarity, with some significant or interesting variations.

Mosher [5] reported on 13 cases of slag burns of the aural canal and tympanic membrane seen in acetylene welders. He discussed thoroughly the symptoms and treatment and alluded to similar cases reported by Lion [6] and by Heermann.[7] Britton and Walsh [8] mentioned the occurrence of otitis media following perforation of the drum head by molten metal, and, finally, McNaught [9] stressed injury to the aural canal and tympanum from slag as a new industrial and potential medicolegal hazard. Simonton [10] stated that gasoline in the aural canal causes severe pain and that if the

gasoline is promptly removed damage is limited to acute inflammation of the canal and drum head. Prolonged exposure to the irritant may cause necrosis of the tympanic membrane. Taylor [11] reported on the spread of nasal and aural screwworm (Cochliomyia americana) infestation in some of the southern states, and Baker [12] recently recorded a case of invasion of a normal aural canal by a green-bottle fly worm (Phaenicia sericata).

Markoff [13] reported an interesting case in which a worm had invaded the ear canal and penetrated the tympanic membrane, simulating a pulsating discharge from the middle ear; and Seshachalam [14] recorded the rare case of a live scorpion in the auditory canal. Foster,[15] Mosher,[5] Zappala,[16] and others described cases in which a wood tick was found in this area, and Zappala [16] believes that his case of a leech in the external auditory canal is the first to be reported in the literature. A patient of Piquet and Decroix [17] was stung in the external auditory canal by a spider. Extraction was difficult and the tissue reaction severe.

Foreign bodies reach the external auditory canal by act of the person concerned or by other persons, accident, or carelessness. In some instances penetrating foreign bodies may enter the canal through the periauricular tissues. Ochs [18] reported an unusual case in which a pipe stem reached the tympanum and aural canal via the oral cavity, and in Hempstead's [19] Case 3 a bullet reached the ear canal and middle ear from a point of entrance to the right of the nose. Having reached the sanctuary of the auditory canal, foreign bodies may remain in situ or lodge in the surrounding structures as a result of their own momentum or unskilled attempts at removal. Simonton [10] stated that, in addition to the trauma incident to removal, the tympanic membrane may occasionally be injured by foreign bodies as a result of direct trauma due to the foreign body, caustic or irritating action of the foreign matter, or prolonged contact of the foreign body with the drum membrane. Warren and Bivins [20] reported two cases of a knife blade lodged in the bony canal wall. In one instance the point of the blade penetrated the petrosa, and in the other instance the point was embedded in the mastoid process. In Proctor's [21] patient a bullet was found in the floor of the canal. In Mosher's [5] case a knife blade entered the canal anterior to the ear, transversed the canal, and the tip lodged in the middle fossa of the skull in close proximity to the jugular bulb. Bettington [22] recorded a case of a pussy willow catkin which entered the aditus through the aural canal. Longaker's [23] patient had a piece of a glass button and a pledget of cotton in the mastoid as a result of numerous previous unsuccessful attempts at removal from the external auditory canal. In Wood's [24] case a part of a scissors blade traversed the ear canal and lodged in the medial wall of the tympanum as a result of a freak accident. This case also possessed medicolegal potentialities.

The symptoms of foreign bodies in the external auditory canal depend to a large extent on the nature of the foreign body, location in the canal, and length of sojourn. There may be a complete absence of symptoms in many cases, while in others varying degrees of pain, discomfort, impaired hearing, discharge, tinnitus, itching, and bleeding may prevail. Symptoms may develop immediately upon lodgment of the foreign body in the canal or only after a latent period of weeks, months, or even years. Politzer [25] removed a piece of slate pencil 3 cm. long from the ear of a patient who knew of its presence for 50 years. There were no symptoms until a plug of cerumen caused deafness. He also removed from the ear of one of his students a piece of lead pencil 1 cm. long, which had been present for 22 years without symp-

toms. He, moreover, referred to asymptomatic aural foreign body cases of long sojourn, as reported by Brown (pebbles in the ear canal for 7 years); Lucae (cherry stone in the ear for 40 years); Zaufal (cherry stone in the ear for 42 years); Reim (molar tooth in the ear for 40 years); Maschal (a coral rosary bead present in the ear 45 years); Marian (glass pearl in the ear for many years). Additional similar cases are referred to by Toynbee,[26] Piquet and Decroix,[17] and Cruz.[27] The latter author referred to a case reported by Wintergothan in which a cherry seed remained asymptomatic in the ear canal for 70 years. Wells [28] recorded the unique case of a pea lodged in a patient's ear for 33 years without the patient's knowledge of its presence. Achaval [29] and Tapia [30] discussed the symptoms and other phases of foreign bodies in the external auditory canal in a very comprehensive manner.

In cases recorded by Warren and Bivins,[20] Mosher,[5] and Hempstead [19] suppuration incident to foreign bodies in the aural canal was delayed for long periods of time. In Hilton's [31] case a portion of a pencil eraser previously unsuccessfully extracted remained in the ear canal for four years before infection supervened. In Roberts' [32] patient a cork remained in the aural canal for several years before giving rise to pain and infection. It resembled a sequestrum. Lowry's [33] patient, a 40-year-old woman, had been deaf all her life. The author removed a cotton plug embedded in cerumen from the ear canal, and hearing was immediately restored in the affected ear.

Certain symptoms of a reflex nature may develop as a result of foreign bodies in the aural canal. Piquet and Decroix [17] discussed the early literature on this subject and tabulated these reflex symptoms along with the reporting authors as follows: coughing (Arnold, Bowen, Politzer, Kennedy); vomiting (Arnold, Kiesselbach); violent headache (Bompet) occasionally with vertigo (Chavanne); pain in the temporal region or in the teeth (Brieger, Urbantschitsch), and, finally, epileptic crises (Kupper and Schurig). The case reported by Chavanne [34] is of particular interest, since his patient had been subject to headache and vertigo for 20 years. General medical treatment had been prolonged, and the patient had been exempt from military service because of weakness and anemia. During this time no ear examination had ever been made. Headache and vertigo were relieved immediately when the author removed a bead and a button from the ear canal. Abdulla [35] reported a case of a cotton plug embedded in cerumen, which was responsible for persistent hiccough for 19 days, and in Kennedy's [36] patient a grain of rice encapsulated in the auditory canal caused a chronic cough for 36 years. Removal of the foreign body gave immediate relief from cough.

Infections following the presence of foreign bodies in the external auditory canal are relatively rare if the infections incident to unskilled attempts at removal are excepted. Of the complications which do develop, the two most likely to appear are chronic infection and impaired hearing. If the tympanic membrane has been injured as a result of the foreign body, then infection of the tympanum and mastoid may supervene. Foreign bodies which have traversed the external auditory canal in whole or in part and have come to lie in the tympanum or mastoid may occasionally cause injury to the facial nerve, bleeding, or serious intracranial complications. In Foster's [15] case of a wood tick embedded in the soft tissues of the aural canal, there was an associated and unexplained facial paralysis, which cleared up promptly on removal of the tick. Chalier and Rousset's [37] 3-year-old patient succumbed to tetanus,

which developed as a result of an unremoved stone in the ear canal. Machado's[38] adult patient developed tetanus as a result of injury to the aural canal by a twig but recovered from the illness.

Older textbooks on otology lead one to believe that serious and even fatal complications were unfortunately too common after unskilled attempts at removal of aural foreign bodies in the early days of this specialty. Piquet and Decroix[17] stated that Denker collected 20 published cases of fatal accidents occurring under these conditions. The nonexistence of reports of this type in the modern medical literature is a silent tribute to the improved management of these cases.

The diagnosis of foreign bodies in the external auditory canal is made from the history, examination, and x-ray studies in some cases. The history of the presence or absence of a foreign body in the ear is not always reliable. Examination may be hindered by an uncooperative patient, local discharge, bleeding, and stenosis, hyperostosis, or exostosis of the canal. Hara [39] removed exostoses from the canal of a patient and found an unsuspected fruit seed which had been trapped by the bony growths. Its presence was unknown to the patient, and its estimated duration of sojourn was 8 to 13 years. The only possible symptom it could have caused was an occasional snapping sound complained of by the patient. Wood's [24] report is a classic example of the value of x-rays in selected cases and illustrates medicolegal potentialities. In cases of very small animate or inanimate foreign bodies lodged in a deep tympanic sulcus, the diagnosis may be arrived at only after thorough syringing of the ear. The case reported by Reis [40] is an example of how difficult the diagnosis of a foreign body in the aural canal may be at times.

The treatment of foreign bodies in the ear canal consists in their removal with or without a general anesthetic. A variety of forceps, hooks, spoons, and curettes have been devised for this purpose, all of which are safe enough if skillfully used. Syringing is, perhaps, most universally employed for this purpose. It is contraindicated in the removal of foreign bodies which tend to swell, such as vegetables, and it has limitations when there is an associated large perforation of the tympanic membrane. In the latter instance the irrigating solution enters the perforation, and since the reflecting surface of the drum membrane is lost, the force of the irrigating solution is largely dispelled. Vegetable foreign bodies which have become swollen may first be dehydrated by alcohol as a preliminary step to removal. The agglutination method of removing foreign bodies is an old one, applicable in a limited number of cases. It was recently revived by Dixon,[41] who used dental cement on a camel's hair brush to remove a cherry stone. Mosher [5] removed a piece of metal slag from the middle ear via the aural canal by means of a magnet and curved probe.

Animate foreign bodies are best removed by syringing after preliminary anesthetization with ether or drowning with oil or a chloroform and oil mixture, as suggested by Richards.[42] Maggots should first be rendered lifeless by chloroform (as a vapor blown into the ear or as a solution dropped directly into the ear), after which they should be removed by syringing and forceps.

Foreign bodies which cannot be removed through the canal must be removed surgically. The postauricular approach for this purpose was suggested by Paul of Aegina and has long been the approach of choice. Von Troltsch [43] advocated an incision above the ear in children for the removal of foreign bodies deep in the canal. He contended that because of the small bony canal and the sinking-in of the temporal bone at this point the auditory canal is easily separated from the squamous portion

of the temporal bone. A large obtuse angle leading to the drum membrane results, thereby facilitating introduction of instruments for removal. Damage to the postauricular artery is avoided. Proctor [21] removed a bullet from the floor of the bony ear canal via the endaural approach and recommended its use for this purpose, as have others.

The present series of foreign bodies in the external auditory canal consists of 48 patients with 52 foreign bodies. The right ear was involved in 28 cases and the left ear in 18. The condition was bilateral in two patients. One patient was twice a host to a foreign body, and an additional patient presented three unsuspected foreign bodies, two in one canal and one in the opposite canal. The series included 32 males and 16 female patients, of whom 35 were white and 13 Negro. There were 15 animate and 37 inanimate foreign bodies. The age distribution of the patients is recorded in Table 1 and the character of the foreign bodies in Table 2. In 22 cases the presence of the foreign body was unknown to the patient. Eleven of these were adults, and 11 were children, the oldest of which was 11 years of age.

TABLE 1.—*Age Distribution of Patients with Exogenous Foreign Bodies in the External Auditory Canal, Nasal Cavity, and Esophagus*

Age Group, Yr.	Patients, No.		
	Aural Canal	Nasal Cavity	Esophagus
1- 5	8	16	20
6-10	11	1	2
11-15	7	0	1
16-20	3	0	2
21-30	3	0	1
31-40	8	0	0
41-50	6	0	5
51-60	1	0	2
61-70	1	0	4
71-80	0	0	1
Total	48	17	38

In Politzer's [25] experience 90% of his cases of foreign bodies in the ear canal had previous attempts at removal. Rosenwasser [44] and others have had the same experience. In this series 13 patients fell into this category. In one case each camphor and castor oil had been poured into the ear in an attempt to dislodge the foreign bodies. In two cases a physician had been unsuccessful in his attempts at removal, and in an additional case the mother, father, and physician had failed in their efforts at extraction. In the remaining cases an unsuccessful attempt at removal had been made by patients, parents, or relatives employing such common articles as tweezers, match stick, bobby pin, straight pin, wire, or syringe. With one exception previous attempts at removal did not add to the difficulty of my own treatment, since efforts at removal were not persisted in when efforts at the home or physician's office were not immediately successful.

Nine patients in this series presented trauma to the ear canal, tympanic membrane, or both as a result of the action of the foreign body, attempts at removal, or both. In one case there was perforation of the tympanic membrane and excoriation of the auditory canal as a result of the action of the foreign body alone. In one case there was a perforation of the tympanic membrane only and in two cases excoriation of the aural canal only as a result of the action of the foreign body. In three patients

there was excoriation of the canal and in two cases excoriation of the canal and hemorrhage into the drum membrane as a result of previous attempts at removal. In both instances the perforations healed promptly. No significant infection supervened in any case, with the exception of one in which this point is not known.

Out of 48 patients with foreign bodies in the external auditory canal, 47 were removed in the office without anesthesia. One uncooperative child seen at night was hospitalized and etherized in order to remove the foreign body safely. The usual method for removing animate foreign bodies consisted in preliminary anesthetization by holding a cotton-tipped applicator soaked with ether at the auditory meatus for several minutes followed by syringing. Most inanimate foreign bodies were removed by means of a dull ring curette or forceps, supplemented occasionally by syringing.

TABLE 2.—*Character of 52 Exogenous Foreign Bodies in the External Auditory Canal in 48 Patients*

Type of Foreign Body	
Animate	No.
Small winged insects	8
Small bugs	4
Large beetles	2
House fly	1
Total	15
Inanimate	
Stick, match head, match stem (each 1)	3
Solidified sulfa crystals (bilateral, 1 patient)	3
Pencil graphite	4
Pencil rubber	1
Cotton	6
Fruit pit	1
Paper	2
BB shot	3
Beads	3
Beans	2
Chain link	1
Rocks	2
Glass (1 patient)	2
Wheat (1 patient)	2
Solidified castor oil	2
Total	37

In no instance was a significant amount of trauma imparted by these methods. Small children were restrained by pinning them in a heavy cloth made for this purpose.

In this series of 48 patients there were 30 who had no aural symptoms referable to the foreign body. Of this total of 30 patients the presence of the foreign body was known in 9 and unknown in 21 cases, the latter total including 1 patient who was twice a host to a foreign body. In the 21 cases in which the presence of the foreign body was unknown, 17 of the foreign objects were inanimate and 4 animate in nature. In the latter group three animate foreign bodies were adherent to a small mass of cerumen and one caught in the drainage of a chronic running ear. In one additional patient the presence of a foreign body was unknown, but deafness was present as a symptom because of the foreign body. In the nine patients with known foreign bodies but no symptoms, the foreign bodies in all instances were inanimate in nature.

In 18 cases in which the presence of a foreign body was known and symptoms were produced, 8 of the foreign bodies were inanimate and 10 animate in nature. In

the inanimate group pain was the predominating symptom in four patients. It was the only complaint in one of these. In two of these patients pain and bleeding were present and in one pain and tinnitus. Two patients had the single complaint of deafness, and one each complained of a rattling or moving sensation. The 10 patients with animate foreign bodies presented a much wider variety of symptoms than those patients with the inanimate foreign objects. Two patients complained of a fluttering sensation alone, one patient had fluttering and pain, and one patient each complained of discomfort or a tickling sensation. In the remaining six cases there were a variety of symptoms present, of which a fluttering sensation was combined with at least two other complaints, such as scratching, discomfort, pain, bleeding, impaired hearing, itching, and tinnitus. Two patients of this group presented reflex symptoms, such as dizziness, nausea, sweating, faintness, and weakness.

Report of Cases

Case 1.—R. D., a white boy, aged 15 years, was first observed March 27, 1948. The patient was an epileptic, who had been fishing on the bank of a small creek a few hours previously. He had a grand mal seizure, and on recovery he noted a severe sharp pain and roaring tinnitus in the left ear. Examination revealed a fresh blood clot and a portion of an old twig in the left ear canal, with excoriation of the floor and a large posterior-central perforation of the tympanic membrane. A. S. hearing was 15/15 for whispered voice. Treatment: Foreign body removed with forceps; blood aspirated; sulfonamides given orally and penicillin by injection. No infection developed. Perforation completely healed April 23, 1948. He was last observed Feb. 13, 1950. Left tympanic membrane slightly thickened and retracted. A. S. hearing was 15/15 for whispered voice.

Case 2.—G. M., a white man, aged 20 years, was first observed April 16, 1949. The patient was awakened at 1 a. m. by pain, buzzing, impaired hearing, and slight bleeding from the left ear. When the buzzing was intense the patient became quite dizzy and felt very weak. Examination revealed blood in and excoriation of the aural canal, with a bug in contact with the tympanic membrane. Treatment: etherization and syringing.

Case 3.—K. J. S., a white girl, aged 4 years, was first observed Oct. 18, 1950. The father of the patient gave the following history on initial inquiry: While lying on the bed at home, the patient put a BB shot in her left ear and informed the parents of this fact. There were no symptoms. The mother tried unsuccessfully to remove the shot with a bobby pin. The child was then taken to the family physician, who also made an unsuccessful attempt to remove the foreign body and then advised the care of a specialist.

Examination revealed bleeding from the left ear, which the father attributed to the physician's efforts at removal. A blood clot was removed from the ear canal by suction, with immediate increased bleeding, which obscured the field and hampered vision. Approximately 30 minutes was required to control hemorrhage by frequently applied epinephrine packs, pressure, and suction. During this time it was noted that the bleeding was coming from a laceration on both the anterior and posterior walls of the canal at points approximately equal to each other and lateral to the isthmus. The impression was gained that the injury came from the physician's efforts to remove the shot with forceps.

After bleeding was controlled the BB shot was clearly seen lying against the tympanic membrane, from whence it was quickly and easily removed with a dull ring curette, the child remaining remarkably tolerant during the entire procedure. There was no injury to the drum membrane.

While the foreign body was being examined, the father then volunteered the following significant addenda to his previous history. After the mother's failure to remove the shot, the father conceived the idea of inserting a pair of closed sharp-pointed household scissors into the ear canal and then opening them in an attempt to "open up the ear so the shot would roll out." At the same time he inclined the child's head to the left. The foreign body obviously was not removed by this maneuver, and the parents, becoming frightened at the profuse bleeding

which ensued, sought the aid of the family physician. It developed that the latter's attempt to remove the shot was limited to a few efforts to cleanse the canal with cotton applicators. The patient failed to return, and the sequelae are unknown.

Case 4.—M. J., a Negro girl, aged 8 years, was first observed Jan. 10, 1952. The patient was standing sideways at the blackboard at school, and while awaiting her turn to recite she stuck a sharpened pencil into her right ear canal. A visiting teacher unexpectedly opened the door adjacent to the blackboard, the back of the door striking the pencil and driving it deep into the ear canal. There was immediate severe pain and profuse bleeding. Examination in the office a short while later revealed extensive laceration of the anterior wall of the ear canal lateral to the isthmus. The broken piece of graphite was lying free on the floor of the canal and was removed with forceps. There was no injury to the tympanic membrane or further undue bleeding. Treatment: Oral administration of sulfonamides and penicillin, with a further uncomplicated course.

Case 5.—T. K., a white boy, aged 11 years, was first observed in the office Jan. 19, 1953. The patient was referred for opinion as to the advisability of a tonsillectomy and adenoidectomy. Examination revealed the presence of a large piece of graphite from a pencil in the left ear and a pencil eraser and a small beetle in the right ear. Their presence was unknown, and there were no symptoms. Treatment: removal with forceps.

Case 6.—M. L. S., a white man, aged 43 years, was first observed April 23, 1953. While en route to town a few hours earlier, the patient's car skidded on the pavement and overturned several times. He was covered by shattered glass from the windshield of his car but escaped injury except for minor bruises. The patient was brought to town and while walking along the streets, he became conscious of a moving sensation in his right ear. Examination revealed two triangular pieces of glass resting against the right tympanic membrane, with apex up and base down, one fragment being anterior and the other lying posterior to the handle of the malleus. There was no injury to the canal or tympanic membrane. Treatment: removal with cotton-tipped applicators coated with petrolatum (agglutination).

Case 7.—R. C., a white boy, aged 12 years, was first observed June 20, 1953. The patient stated that two days before, while he was playing in a bin of wheat at a nearby orphanage, a companion doused his head deep into the grain. After leaving the bin, the patient removed one grain of wheat from his left ear. Since the incident he had been completely deaf in his left ear and reported this fact to his matron. Examination revealed a grain of wheat lodged in the isthmus of the left ear canal, and there was a total loss of hearing for whispered voice in this ear. Treatment: One grain of wheat was removed with a ring curette, and inspection then revealed a second grain of wheat, with the flat surface in contact with the anterior half of the tympanic membrane. This was removed with a ring curette, and the hearing for whispered voice was found to be normal. The tip of the grain of wheat lodged in the isthmus of the canal apparently contacted the convex surface of the grain in contact with the drum membrane, creating an effective tamponade of the drum head and loss of hearing.

Comment.—Loss of consciousness and automobile accidents are not common causative agents for foreign bodies in the external auditory canal. Sulfonamides administered orally and penicillin orally or by injection seemed to be of definite value in preventing secondary infection in those cases with trauma to the external auditory canal, tympanic membrane, or both. Case 3 illustrates how unskilled attempts at removal of a foreign body in the ear canal may cause more damage than that wrought by an innocuous foreign body. Case 2 is of interest because of the reflex symptoms produced and Cases 6 and 7 because of the unique mode of entry of these foreign bodies into the ear canal.

EXOGENOUS FOREIGN BODIES IN THE NASAL CAVITY

Foreign bodies in the nose, as foreign objects in the external auditory canal, are usually seen promptly, produce few symptoms, and are easily removed. Foreign objects in this area may be animate or inanimate; single or multiple; unilateral or

bilateral. Animate intruders may include a wide variety of insects, maggots, intestinal worms, leeches, and the like. They are seen less frequently in the nasal cavity than in the aural canal.

Foreign substances usually enter the nose through the anterior nares as a result of the action of the person concerned or other persons, carelessness, or accident. Patients include children, adults, and mentally normal and abnormal persons. In traumatic cases portal of entry may be gained through the soft or bony tissues of the nose or via the adjacent orbit, antra, ethmoidal sinuses, or superior maxilla. Entrance to the posterior nares may be gained through the nasopharynx as a result of vomiting, eructations, or sudden sneezing or coughing during the act of swallowing. Paralysis or loss of the palate predisposes to this mode of entry. Maggots are found in the nose as a result of growth and development. Most foreign bodies found in the nasal cavity lie between the inferior turbinate and septum, occasionally between the middle turbinate and septum, and rarely in the meatuses.

Wolfe [45] reported a case in which a knife blade driven through the dorsum of the nose lodged in the floor of the nasal cavity for nine years before being removed. Infection was present, but there were no bony changes. Trexler [46] recorded a case of a large piece of glass found in the floor of the right nostril, which was introduced through the right cheek, upper lip, and nasal vestibule area as a result of an automobile accident seven years before. Prior nasal operations had been performed and treatment for chronic nasal infection rendered by others without the foreign body being suspected or found. In Kelemen's [47] case a splinter of wood (from the tip of an umbrella) introduced into the right nostril during a fall was carried for 28 years in this location before it finally ulcerated through the entire lateral nasal wall and was spontaneously extruded through the right cheek. In Cantoni's [48] case a pencil stabbed into the left orbit penetrated the left ethmoids and nasal septum, coming to rest in the floor of the right nasal cavity posteriorly. The only symptoms were a persistent draining sinus of the left conjunctival sac and granuloma and nasal infection of four months' duration. In Howard's [49] patient a knife blade entered the left zygomatic bone and penetrated the left antrum and nasal cavity, finally coming to rest in the hard palate beneath the right nostril and right antrum. In the case reported by Voorhees [50] a metal bolt was introduced into the nasal cavity when a homemade gun exploded in the patient's face. Symptoms of infection developed seven months later, but the foreign body was not removed until 17 years subsequently. In Vadala's [51] patient a knife blade passed through the right maxillary sinus and transfixed the right inferior turbinate, and the point pierced the nasopharynx. There were no symptoms for one year. In all of these cases the presence of the foreign body was unknown to the patient.

Foreign objects such as cotton, gauze, or materials for surgical implants sometimes lodge in the nose as a result of medical or surgical treatment. Dowling [52] reported his own experience in which a sphenoid probe of inferior metal broke while he was sounding the sphenoid sinus. The broken probe was recovered from the inferior meatus. Myerson [53] and Pearlman and Pilot [54] each reported a case in which gauze in the nose produced a clinical picture simulating carcinoma. In the case of the former the reason for the gauze in the nose was undetermined. In the case of the latter it apparently was left in the nose after a previous nasal operation, and a foreign body granuloma resulted. The authors provide a detailed pathological study.

The symptoms of foreign bodies in the nasal cavity depend upon the character, location, size, and duration of the intruder. In the average case of foreign body seen in a child's nose in office practice, the character of the foreign body is such that trauma is minimal, symptoms few, if any, and treatment simple. Sharp or irregular foreign bodies may cause bleeding as an initial symptom. Usually within a few days after lodgment of any type of nasal foreign body, nasal obstruction, mucoid nasal drainage, sneezing, and pain or discomfort appear. The drainage soon becomes purulent, foul, and often blood-streaked, with infection of the nares and upper lip often developing as a sequel to the irritating discharge. Occasionally, symptoms incident to a nasal foreign body may be fulminating. Longaker [23] recorded a case in which a boy placed a paper Fourth-of-July snake in his nose. In four days an acute abscess of the hard palate developed and was incised. Six days later the foreign body was removed from the area between the nasal septum and the right inferior turbinate. A portion of bone in the floor of the nose was necrotic and had to be removed along with friable portions of the right middle and inferior turbinates. An acute right maxillary and ethmoidal sinusitis developed within this interval. In the case recorded by Santos [55] a thread lodged in the nasal cavity of a child caused an acute orbital abscess. The nasal foreign body was unsuspected until late in the course of the disease. It was extruded spontaneously after unsuccessful attempts at removal produced hemorrhage.

Nasal foreign bodies do not always occasion immediate symptoms. A latent period of months or even years may be present between time of lodgment and onset of symptoms. Camerer [56] removed from the right nostril of a patient a partially calcified cork, which had been in place for 17 years. There were no symptoms, or else they were ignored. Altounyan [57] removed a coin from beneath the mucosa of the right inferior meatus of a 20-year-old patient. The coin had been placed in the nose in childhood. The patient's symptom of nasal drainage had been present for one year. Kelemen [47] believes that his case of a nasal foreign body present for 28 years is of unequaled duration and the only one reported in the literature exhibiting spontaneous expulsion by transmigration. In his search of the literature for comparable cases, the following reports were found and recorded: Erdelyi extracted a piece of broken adenotome from the nasal cavity; Kissmann, a vegetable stalk; Baudet and Lo, a splinter of shrapnel. In these three instances the foreign body had been present 20 years. The author stated further that Gelfand's patient carried a piece of iron in his nose for 23 years and Krysing's patient a projectile for 25. In most of these cases there were no symptoms over long periods of time until sudden and acute signs developed. He refers to two cases from the literature in which there was transmigration of foreign bodies to different parts of the nasal cavity.

Symptoms incident to nasal foreign bodies may be present for months or many years without the presence of a foreign substance being suspected by the patient. Most of the cases already cited have been of this nature. Marks [58] removed from between the middle turbinate and septum a twig, which had been in a patient's left nostril 8 to 12 years. Destruction of the nasal bones was present, and carcinoma was suspected. The twig was apparently inserted during a period of sanatorium treatment for involutional melancholia incident to a hysterectomy. Donnelly's [59] 7-year-old patient complained of right nasal obstruction and right frontal headache, with tenderness over the nose for two years. There was no discharge or odor. A partly corroded screw was demonstrated by x-ray and later removed. Clouding of the right

antrum and ethmoids was apparently due to poor ventilation rather than suppuration. Ts'en's [60] 5-year-old patient had a foul nasal drainage, which was disregarded by the parents for 18 months. A leaf was removed from the right nostril.

Symptoms of a general nature sometimes accompany nasal foreign bodies. Magee's [61] 5-year-old patient had such a marked loss of weight and appetite as to simulate tuberculosis. The only nasal symptom was a slight watery drainage from the right nostril. Examination revealed a nasal polyp and a rubber tack, which had apparently been in place about one year. Both were removed, with marked general improvement. Poe [62] reported similar type cases.

Reflex symptoms are sometimes seen with nasal foreign bodies. Steele's [63] 6½-year-old patient had experienced convulsions and mental retardation for two years. Nasal drainage was scant and foul. All treatment had been to no avail. Symptoms were relieved by removing a shoe button from between the middle turbinate and septum. Poe [62] reported asthma and gastroenteritis incident to nasal foreign bodies in children.

Animate foreign bodies sometimes invade the nasal cavity. Brown [64] reviewed the early literature on nasal screwworm infestation. Taylor's [11] report of his personal experience with the symptoms, diagnosis, complications, and treatment of this condition is exhaustive. Wilson's [65] patient presented him with a centipede he had just blown from the nose. The author concluded from the history and symptoms that the centipede must have undergone some period of development in the nasal cavity. Astavans,[66] Khan,[67] Narasimhaiah,[68] and Narayanan [69] each reported a case of a leech in the nasal cavity. In Astavans' case the leech was 5 in. (13 cm.) long and protruded into the nasopharynx. Meyer [70] stated that leeches are a very common type of nasal foreign bodies in Anatolia between June and October. He reported a personal experience with more than 100 cases of leeches in the nose, pharynx, larynx, and mouth. These parasites commonly invade the nose while the patient swims or drinks infested water from streams or water bottles. They commonly attach themselves to the turbinates because of the abundant blood supply. The chief nasal symptoms are obstruction and hemorrhage, the latter sometimes leading to severe anemia. Removal is best accomplished by forceps. In the case reported by Muller [71] a bug lodged in the nasal cavity caused repeated severe hemorrhages.

The commonest complication of nasal foreign bodies is chronic infection of the nose and paranasal sinuses, due to mechanical obstruction produced in the nasal cavity. The ethmoids and antra are particularly vulnerable. Destruction of bone may occur from pressure necrosis, infection, or both. In the case reported by Rao [72] a tamarind seed recovered from an infected right antrum had apparently been placed in the nose and subsequently ulcerated through the lateral nasal wall into the sinus. Koepcke [73] stated that foreign bodies in the nose may invade the antrum via the natural or accessory ostium or by ulceration through the nasal wall. The cherry stone, which he personally removed from the right antrum, may have arrived there by ulceration through the nasal wall or through the alveolar openings of freshly extracted teeth. Brown's [74] patient died of tetanus as a result of a bead placed in the nose seven days before. Burton [75] reported three cases of nasal foreign bodies associated with nasal diphtheria and reviewed the literature on this subject. One case each of his was due to tape from a necktie, a pea, and a shoe button. Canfield [76] mentions in a more recent publication a case of this nature due to a shoe button. Foreign bodies entering the nasal cavity sometimes serve as the nucleus for the formation of

a rhinolith. Polson [77] made an exhaustive study of this subject and reviewed the literature. He stated that up until 1943 about 387 rhinoliths had been reported in the literature, some of which were due to foreign bodies. Rhinoliths sometimes attain huge proportions. In Myerson's [78] case a rhinolith weighing 56 gm. had to be removed by an external operation. Larger ones have been reported.

The diagnosis of nasal foreign bodies is made in most instances from the history and examination. The former may be valueless if the presence of the foreign body is unknown to the patient, and the latter may be complicated by the chronic inflammatory reaction of the nasal tissues and paranasal sinuses associated with the nasal intruder. Nasal polyps may occasionally be associated with and carcinoma simulated by nasal foreign bodies. Pearlman and Pilot [54] reported a case of the former, discussed the histology, and reviewed the literature. Marks,[58] Myerson,[58] and Pearlman and Pilot [54] described examples of the latter. Trexler [46] stressed the value of probing fistulae about the nasal cavity as an aid in diagnosing nasal foreign bodies. X-rays are of value if the foreign object casts a shadow. Kopp and Levy's [79] 6-year-old patient developed frequent colds at the age of 3 years, and one year later he developed bilateral suppurative otitis media. The tonsils and adenoids had been removed, and repeated nasal treatments had been given by other physicians before the authors demonstrated by x-ray the presence of a button in the nasal cavity. It was removed from a position between the septum and the posterior tip of the inferior turbinate with an edge in the middle meatus. Gutherie's [80] 35-year-old patient complained of nasal drainage for 18 years. The tonsils and adenoids had been removed by another physician at the onset, without benefit. The author removed a cherry stone from between the posterior tip of the inferior turbinate and septum. He also reported on several cases of marine sponges as nasal foreign bodies, which he had not seen previously recorded in the literature.

Most foreign bodies in the nose may be removed in the office by means of forceps or dull ring curettes and without the use of anesthesia. Syringing is rarely indicated. In cases in which foreign bodies are large or occupy adjacent accessory sinuses as well as the nasal cavity, intranasal or external nasal surgery alone or combined with a Caldwell-Luc operation or other external surgery may be indicated.

The present series of nasal foreign bodies consists of 17 patients who harbored 18 foreign bodies. The right nostril was invaded in 6 instances, the left in 10, and the condition was bilateral in 1 instance. In 16 cases the foreign body was lodged between the inferior turbinate and the septum. In one case the foreign body occupied the space between the inferior and middle turbinates and septum in each nasal cavity. There were nine male and eight female patients, all of whom were white. There was a total of 18 inanimate foreign bodies. The age of the patients is recorded in Table 1. As to the character of these 18 foreign bodies, there were two cases of pencil erasers, two butter beans seen in one patient, and one case each of a rubber stopper, hard rubber, crayon, corn kernel, peanut, paper, flower petal, berry, leaf, coal, button, cracker, chewing gum, and candy. In 6 patients the presence of the foreign body was unsuspected by the parents, and in the remaining 11 the presence of a foreign body was known or suspected by the parents.

In five cases previous attempts to remove the foreign body had been made by a parent, physician, or both. There was no trauma in any of these cases that might be attributed to the foreign body or attempts at removal, but in all instances attempts at removal had only succeeded in pushing the foreign body further in the nasal cavity.

In all cases the foreign bodies were removed in the office without anesthesia and with or without restraint, employing forceps, a dull ring curette, and occasionally suction.

In 8 of the total of 17 patients with nasal foreign bodies there were no symptoms referable to the foreign body. These were all cases in which the foreign body had been in situ only a few hours. In the remaining nine cases there was one case each with the complaint of watery nasal drainage, nasal obstruction, and bleeding. In all of the other six cases there was complaint of foul purulent nasal drainage with blood streaking or bleeding, and in three of these cases there was infection of the nares and upper lip. There were no symptoms of a reflex nature in any case, nor was there aural or eye pathology associated with any case. All cases in which the presence of the foreign body was known or suspected had been in situ less than six hours. Symptoms of a general nature were present in one patient.

Report of Cases

Case 8.—C. K., a white boy, aged 7 years, was first observed Dec. 10, 1948. Nine months previously the patient developed from the left nostril a purulent nasal drainage that soon became foul and frequently was blood-streaked. The left nostril became sore and chronically irritated. Two months after onset of the nasal discharge the patient developed recurrent attacks of acute tonsillitis, which were resistant to treatment. On one occasion a peritonsillar abscess developed and was drained. On several occasions the referring physician cleansed and examined the nostrils and prescribed nose drops, with temporary improvement in drainage and odor. He was unable to see a nasal foreign body. When nasal treatment finally produced no results, and because of the constant permeating stench, the patient was referred for diagnosis and treatment.

Examination revealed a large pencil eraser wedged between the anterior third of the left inferior turbinate and adjacent nasal septum. The turbinate was edematous and overlapped the eraser in a cap-like manner. A few granulations were present on the inferior turbinate and adjacent septum. There was a small amount of foul pus present, and the left anterior naris was swollen and crusted.

Removal of the foreign body with forceps was attended by brief bleeding, which was easily controlled. Two weeks later the nose was healed.

Case 9.—D. C., a white girl, aged 3 years, was first observed Jan. 10, 1951. Three days before the mother of the patient noted a foul odor about the child's person and thought that a bath was in order. This failed to alleviate the odor, and the baths were repeated frequently on the same and following day and with increased vigor of scrubbing. The source of the odor at this time was a mystery. On the third day a foul drainage developed from the right nostril, and the child was thought to have an abscess in the nose. On the morning of Jan. 10, 1951, the right nostril bled freely at home. Medical attention was sought because of the bleeding.

Examination revealed a large wad of blotting paper between the anterior third of the right inferior turbinate and the adjacent septum, with intense congestion in these areas. There was a mild infection of the right nasal vestibule.

Removal of the foreign body with forceps was attended by bleeding, which was easily controlled.

Case 10.—L. F. J., a white girl, aged 2 years, referred by Dr. E. Tucker, was first observed Sept. 10, 1951. For the past two months a bilateral purulent nasal discharge had been present, associated with nasal obstruction, difficulty in nursing a bottle, restlessness, irritability, insomnia, noisy breathing at night, and weight loss. During the past three or four weeks the drainage had been foul, and on one occasion there had been mild bleeding from one nostril. At the first visit to the referring physician he prescribed nose drops. When there was no improvement three days later, he inspected the nasal cavities and, noting the presence of some type of foreign body, referred the patient for treatment.

Examination revealed a large butter bean wedged between the nasal septum and the middle and inferior turbinates in each nasal cavity. There was profuse foul pus present, and the nasal

mucosa was intensely congested. No granulations were present. The foreign bodies were removed in the office with forceps with some difficulty. Recovery from both local and general symptoms was rapid.

CASE 11.—E. F., a white girl, aged 2 years, referred by Dr. O. W. Cranz, was first observed June 6, 1952. About one week before, the patient's left nostril began to bleed during sleep with spontaneous arrest. Several days later a thin, foul purulent drainage developed. The mother of the patient inspected the nose and, seeing "proud flesh," sought medical advice.

Examination revealed a small button in close contact with the left Kiesselbach's area. Foul pus was present but no granulations. The foreign body was removed with forceps.

CASE 12.—W. J., a white boy, aged 3 years, was first observed Dec. 30, 1953. Two months before, the patient developed a "bad breath," the cause of which defied detection by the parents. Soon afterward the left nostril began to bleed mildly at intervals. Recently there had been purulent nasal drainage from the left nostril and soreness in this region.

Examination revealed a pencil rubber between the nasal septum and inferior turbinate on the left side, with small granulations present. Foul pus and infection of the left anterior naris were associated.

Removal of the foreign body with forceps was attended by rapid improvement when the patient was last seen Jan. 3, 1954.

Comment.—It is of interest to note that in Cases 9 and 12 the initial symptom of a nasal foreign body was foul odor of undetermined origin and location. Case 8 demonstrates that the diagnosis of nasal foreign bodies producing textbook symptoms may still be missed. Case 10 shows that nasal foreign bodies may produce bilateral as well as unilateral foul purulent nasal drainage, thereby complicating the diagnosis. The degree of swelling which the beans underwent in this case must have been tremendous, since they were extracted with difficulty and since the bean could not be reintroduced into an anterior naris. Nasal foreign bodies may occasionally give rise to symptoms of a general nature.

EXOGENOUS FOREIGN BODIES IN THE ESOPHAGUS

Reports of foreign bodies in the esophagus appear more frequently in the early medical literature than do reports of foreign bodies in the air passages. Clerf [81] attributes this to failure to recognize foreign bodies in the air passages because of inadequate diagnostic methods. The results of improved diagnostic methods, however, are already apparent in the work of Killian [82] in 1907. As foreign objects in the external auditory canal and nasal cavity, foreign bodies in the esophagus are usually seen promptly after the accident. Unlike foreign bodies in these regions, however, the majority of esophageal foreign bodies produce immediate symptoms and are surely more of a problem to treat.

While the variety of foreign objects that may lodge in the esophagus is virtually endless, the majority of the intruders found in this region are coins and disks, bones, food, safety pins, and dentures.* New variants of these general types of foreign bodies may, however, appear on the scene from time to time and often in an interesting manner. Marmor [98] reported on 31 cases in which the Missouri sales tax tokens had been swallowed. Of 13 esophageal foreign bodies removed by Hua Liu,[99] five were date stones seen in a period of two months, when no similar foreign body had been seen in the previous 10 years. Foreign objects may be found in the normal or abnormal esophagus and in the mentally normal or abnormal person of almost any age group.

* References 82 and 83 to 97.

The etiologic synergy of foreign bodies in the normal esophagus of mentally sound patients includes primarily such factors as carelessness, age, poverty, emotions, and unconsciousness from any cause.† Rarely one sees esophageal foreign bodies as a result of a prank.[101] Pathological conditions of the esophagus which favor impaction of foreign objects include such intraesophageal conditions as stricture, spasm, malignancy, diverticulum, and varicosities, and such extraesophageal conditions causing compression stenosis as goiter, aneurysm, etc. Paralysis of the esophagus favors both esophageal and bronchial lodgment of foreign bodies.[94] Holinger and his associates [102] stated that three types of congenital anomalies of the esophagus may be responsible for lodgment of foreign bodies in a part other than the usual cervical portion. These are congenital esophageal stenosis, a congenitally short esophagus, or congenital atresia of the esophagus with a tracheoesophageal fistula, the repair of which has been followed by formation of a stricture at the point of anastomosis. Esophageal foreign bodies are seen in such abnormal persons as the mentally deficient, insane, exhibitionists, would-be suicides, and prisoners. They may be multiple and are sometimes of bizarre nature.‡

The symptoms produced by foreign bodies of the esophagus vary. In most instances the initial symptoms may include choking, gagging, retching, vomiting, discomfort, pain, dysphagia, and odynphagia of varying degrees and duration. Bleeding may occur if there has been laceration of the oral cavity, pharynx, or esophagus. Dry cough of a mild degree and short duration is often present as an initial symptom.

After the initial symptoms have subsided there may be a total absence of symptoms for varying intervals in some cases. Boyd [84] reported that 35 patients with esophageal foreign bodies which were removed within two weeks had no symptoms. These cases included 17 coins, 11 safety pins, three thumb tacks, one cogged wheel, two jacks, and one metal puzzle. Randall [109] reported a single case of a 10-month-old child who was seen to swallow a safety pin. The pin lodged point down in the esophagus, and during six days of observation there were no signs or symptoms.

Dysphagia is a common symptom persisting after the initial symptoms of foreign body ingestion. Odynphagia is not uncommon, and in certain instances aphagia may be present. Chamberlin [110] recorded an interesting case of septicemia with dysphagia so marked as to simulate an esophageal foreign body.

Vomiting is a characteristic symptom and may occur spontaneously once, at intervals, or only after intake of liquids or solid foods. Some patients are able to take liquids without difficulty but vomit solids. Vomiting in small children may be misinterpreted as regurgitation due to "upset stomach."

Discomfort or pain is frequently present after ingestion of a foreign body. The former has been described to me by older children and adults as a fullness, lump sensation, or mild aching and has been present about as frequently as sharp pain. Pain as a symptom of esophageal foreign body may be produced by impaction of a very large foreign object, by penetration of the esophageal wall, or by resulting secondary inflammation. It may be constant or appear only on swallowing. Since most foreign bodies lodge in the cervical esophagus, this is the most common site of discomfort. It is usually localized to the front or either side of the neck. Foreign bodies occupying the midesophageal region cause discomfort or pain which is

† References 94 and 100.

‡ References 103 to 108.

referred to the back. It is known, however, that in some instances the pain referral of a foreign object in the esophagus may be remote from the site of impaction and therefore misleading.§

Increased salivation is not uncommon in adults and children, and drooling is prone to occur in the latter.

Small children who cannot talk because of age or who will not talk after swallowing a foreign body may manifest a complete loss of appetite by refusing to take anything by mouth. When this occurs abruptly during a meal one should be suspicious of a foreign body even though choking or gagging be minimal or perhaps completely absent.

Symptoms referable to the respiratory tract may be present at the time of ingestion, after the foreign body has lodged in the esophagus, or both.‖ Respiratory symptoms such as cough, wheezing, dyspnea, or cyanosis may be present because of the size, shape, position, or other characteristics of the foreign body, and they may completely replace or dominate the usual symptoms accruing from an esophageal foreign body. In studying the symptoms produced by esophageal foreign bodies which had been in situ for one month to four years, Boyd [84] found that respiratory symptoms predominated in 50% of 22 cases.

According to Jackson and Jackson,[118] there are five ways in which pulmonary symptoms may be caused by a foreign body in the esophagus. The presence of a large obstructive foreign object in the esophagus prevents drainage from the nasal and oral cavities from escaping into the stomach as it normally does via the patent esophagus. These secretions overflow into the tracheobronchial tree, with irritating effect and attendant symptoms. Compression stenosis of the trachea may cause dyspnea and a stridorous or wheezing respiration simulating other diseased states. The inflammatory zone surrounding an irritating esophageal foreign body may extend to the upper orifice of the larynx and thence into the endolaryngeal tissues. Foreign objects in the esophagus of prolonged sojourn may ulcerate into the trachea or bronchi, with resulting fistula formation.

The physical signs of esophageal foreign bodies that may be elicited by clinical examination are few in number. Compression of the airway may give rise to previously mentioned respiratory signs. Palpation of the neck may reveal local tenderness. Excessive salivation, with frequent expectoration in adults and drooling in children, may be noted. On examination with the mirror excessive secretions, with or without blood tinge, may be observed in the pyriform sinuses when there is marked obstruction to the esophagus. Friedberg [121] called attention to head inclination in children with esophageal foreign bodies. Jackson and Jackson [85] also described this sign and called attention to the tendency of some children with esophageal foreign bodies to assume a prone position in bed. At esophagoscopy the character of the foreign body, the reaction of the esophageal tissues, or usually both may produce a clinical picture simulating carcinoma. Weaver and Moersch [122] made such an observation in the case of a patient with congenital anomalies of the esophagus, which became obstructed with persimmon skins.

The diagnosis of esophageal foreign bodies may be established after careful evaluation and correlation of the history, symptomatology, signs, roentgen study, and

§ References 111 and 112.

‖ References 113 to 120.

esophagoscopic exploration. The common failure to make a diagnosis is due to failure to consider the possibility of a foreign body or refusal to consider the history. A reliable history of initial choking and gagging in a patient of any age group should be considered as indicative of an esophageal foreign body until proved otherwise. Negative symptomatology and negative x-rays after such a history should be disregarded. X-rays are an invaluable diagnostic aid in determining the presence, character, and localization of opaque foreign substances. Routine anteroposterior and lateral views should be secured from the nasopharynx to the ischial tuberosities and supplemented by additional views if necessary. The use of barium sulfate (as a mixture or as a capsule) or an opaque oil to determine the presence and location of nonopaque foreign bodies is invaluable at times. Iglauer ¶ was apparently the first to employ an opaque oil for this purpose. I have found it more satisfactory than barium. Small bones, and especially fish bones, have always been particularly difficult to demonstrate by x-ray. Goldman [83] and Skarby,[125] however, in recent years have made tremendous progress, both clinically and experimentally, in the demonstration of fish bones in the esophagus by x-ray. The former stated that fish bones are as readily demonstrable by x-ray as are chicken bones.

The pathologic changes produced by foreign bodies in the esophagus are due to obstruction, stenosis, erosion, ulceration, and perforation of the esophageal wall.[102] These pathologic changes set up the mechanism for such complications as obstruction; esophagitis; mediastinitis; cellulitis of the neck; fatal hemorrhage from penetration of a large blood vessel; pulmonary complications, such as pneumonia, abscess, gangrene, or empyema; mediastinal emphysema; pneumopericardium; pericarditis; diverticulae, and stricture.

Partial obstruction to the lumen of the esophagus is a common complication of foreign body lodgment, but complete obstruction is less frequently seen. Esophagitis may range from a mild minimal type to the more serious sloughing variety.

Mediastinitis usually follows perforation of the esophageal wall by a foreign body. While most perforations of the esophagus are due to direct penetration of the wall by sharp-pointed foreign bodies, such as bones, pins and needles, safety pins, etc., in some instances perforation may occur as a result of foreign bodies which produce pressure necrosis.# Coins, large bones and other objects may do this. Latimer [127] reported such an instance in which a radish swallowed by a Mongolian idiot produced pressure necrosis and death. Immediate perforation of the esophagus by a foreign body is not the rule. For this reason the observations of Jackson and Jackson [85] that more than half of the patients who developed serious complications are those in whom foreign bodies have been overlooked by patient, physician, or both is of vital significance. Lerche [97] found that among 200 cases of esophageal foreign bodies reported in the literature between 1900 and 1910 there were perforation and death in 23, or 12%. A review of many more recent publications on esophageal foreign bodies leads one to believe that perforation and death now occur in about 1 or 2% of the cases. Perforation of the esophagus occurs most frequently in the cervical and upper thoracic regions, apparently because these are the most frequent sites of lodgment.

¶ References 123 and 124.

References 97 and 126.

Head [126] classified infections following perforation of the esophagus as follows: (1) periesophagitis consisting of a mild cellulitis subsiding without extension or localization; (2) localized periesophageal cervical abscess—(*a*) small, draining into the esophagus and (*b*) large, encapsulated; (3) mediastinitis—(*a*) acute, spreading as an extension of a cervical cellulitis or as a result of a local perforation and (*b*) localized abscess as an extension from a cervical cellulitis or result of a local perforation.

Laff,[128] Strieder,[129] and Clerf [93] presented evidence that perforation of the esophagus may occur occasionally without clinical evidence of infection. Jemerin and Aronoff [130] reported three cases of esophageal perforation with foreign body migration into the thyroid gland. An abscess of the thyroid occurred in each case, but no paraesophageal infection occurred.

Perforation of a large artery by a swallowed foreign body is not rare. Barrie and Townrow [131] reported a case of perforation of the aorta by an esophageal foreign body and stated that 80 such cases had been reported in the literature from 1840 through 1940. These authors alluded to the report of Schlemmer in 1929, which listed 51 cases of perforation of the aorta, 2 of other vessels in the thorax, and 29 of vessels in the neck collected from the literature. As a result of their review, Barrie and Townrow [131] were able to record some pertinent facts regarding perforation of the aorta by esophageal foreign bodies. The objects responsible for perforation in 39 cases were bones of various types (26), coins (4), safety pins (3), dentures (2), a collar stud, a pin, a needle, and a wire. The interval between swallowing of a sharp foreign body and hemorrhage in 37 cases ranged from five minutes to six weeks, with an average of seven to eight days. Blunt objects required from 14 days to one year and 10 months in which to perforate the aorta. The authors concluded that perforation of the aorta is probably caused by constant pressure, aided by contraction of the esophagus and pulsation of the aorta. Sepsis apparently does not play an important part. Three times as many males as females are affected. A significant feature of the clinical picture of aortic perforation is the so-called "signal hemorrhage" appearing from a few hours to three days before the final fatal bleeding episode.

Other cases of perforation of the aorta have been published since 1940, and large vessels other than the aorta have been perforated by esophageal foreign bodies. Clerf * reported two cases in which the innominate artery and the heart itself were punctured by a safety pin, resulting in the death of the patient. Kearney [132] recorded a case of fatal hemorrhage following a swallowed fish bone which migrated and penetrated the right renal artery. Simons' [133] case report of a fatal hemorrhage from the arch of the aorta due to meat impaction and pressure necrosis represents an interesting medicolegal angle in this type of case.

Important hemorrhage from the esophagus in the absence of major vessel involvement is rare. Moore [134] treated a 14-year-old girl who swallowed a chicken bone. After several weeks there was a fatal hemorrhage, and postmortem showed that the bone was lodged transversely in the esophagus, causing mural necrosis at both ends. Saltzstein [135] recorded a similar case of fatal hemorrhage from a vessel in the wall of the esophagus, due to erosion of the vessel by a duck bone. He referred to the

* References 89 and 93.

report of Poulet in 1880, which listed 33 reports of fatal hemorrhage from esophageal perforation, and only one case was due to rupture of an esophageal artery.

Foreign bodies of the esophagus which perforate into the tracheobronchial tree may cause pneumonia, abscess, gangrene, or chronic fistula. Empyema may develop. Harvey and Davy [136] reported a case in which an esophageal foreign body was responsible for massive collapse of the lung. Mediastinal emphysema may follow a perforation of either the cervical or thoracic esophagus. It is usually associated with subcutaneous emphysema and may prove fatal regardless of whether infection follows the leaking of air, the mechanism apparently being one of shock.†

Arens and Stewart [138] recorded a fatal case of pneumopericardium resulting from foreign objects in the esophagus and stated that only four similar cases had been reported in the literature up until that time. Pericarditis is sometimes seen as a complication of esophageal foreign bodies. Imperatori [139] believes that pericarditis may well be the immediate cause of death in cases of acute mediastinal infection, because of the lymphatic connections between the mediastinum and pericardium. Boyd [84] found that diverticula of the traction type are prone to develop in foreign bodies of long sojourn but tend to recede without surgical therapy after the foreign body has been removed. She also noted the tendency to stricture formation. It would seem, however, that this latter complication is relatively rare, considering the number, variety, and duration of esophageal foreign bodies and the trauma consequent upon their removal.

Less common complications of esophageal foreign bodies include septicemia,‡ osteomyelitis of the cervical vertebrae,[142] and new bone proliferation of the vertebrae.[143]

Cases of foreign bodies in the esophagus of long sojourn are not as numerous as similar cases in the tracheobronchial tree. Perhaps this is due in part to the fact that swallowed foreign objects are not as prone to produce an asymptomatic latent period as are foreign bodies in the bronchi. Medical advice is therefore sought earlier because of persistence of symptoms. According to Jackson and Jackson,[85] patients with esophageal foreign bodies rarely live over five or six years and usually die within one year. The longest sojourn of an esophageal foreign body was recorded by C. L. Jackson.[144] The patient carried a wooden button in the esophagus for 17 years, and, strangely enough, there was no stricture formation incident to its presence. Occasionally carcinoma may develop in cases of long standing.[85] Traction diverticula and stenosis, as noted by Boyd,[84] may develop.

The initial symptoms present in the 38 patients with esophageal foreign bodies in this series have been recorded in Table 3. The symptoms have been arranged in accordance with their dominance of the clinical picture at the time of the accident. Complaint of a severe aching or cutting sensation has been interpreted to mean pain as contrasted to the choking, fullness, or lump sensation of discomfort. Dry cough of brief duration was present in about one-third of the patients. In three patients there were no initial symptoms. The one patient with head inclination was aged 5½ years. The position of the head was noted by the mother while the child was playing, and persistent inquiry finally brought forth a confession from the patient

† References 85 and 137.

‡ References 140 and 141.

that she had swallowed a quarter a short while before. There were no other symptoms. I have observed head inclination as the only symptom in the case of a small child with a fishbone in the tonsil. In an additional small child, head inclination and

TABLE 3.—*Initial Symptoms Present in 38 Patients with Esophageal Foreign Bodies*

Initial Symptoms	Patients, No.
Pain alone	5
Pain, cough	3
Pain, vomiting	2
Discomfort alone	5
Discomfort, gagging	2
Discomfort, gagging, cough	1
Discomfort, aphagia	1
Discomfort, cough	2
Gagging alone	1
Gagging, vomiting, cough	4
Gagging, cough, drooling	2
Gagging, vomiting, cough, cyanosis	1
Gagging, cough	1
Gagging, vomiting	1
Cough, vomiting	1
Loss appetite (refusal to eat)	1
Head inclination	1
No symptoms	3
Unknown	1
Total	38

TABLE 4.—*Symptoms Present at the Time of Examination in 38 Patients with Esophageal Foreign Bodies*

Symptoms	Patients, No.
Pain alone	4
Pain, vomiting	1
Pain, salivation, expectoration	1
Discomfort alone	4
Discomfort, aphagia	1
Vomiting alone	7
Vomiting, restlessness, insomnia	1
Vomiting, discomfort	2
Vomiting, loss appetite, discomfort	1
Vomiting, cough, drooling, loss appetite	2
Vomiting, dysphagia	1
Drooling alone	2
Odynphagia alone	2
Aphagia alone	1
Loss appetite (refusal to eat) alone	1
Gagging, drooling, swallows liquids but vomits solids, hoarseness	1
Swallows liquids, vomits solids	1
Swallows liquids, refuses solids	1
None	4
Total	38

hoarseness were the only symptoms of a large flat fish bone lodged in the subglottic larynx. The patient with the single symptom of loss of appetite was a 19-month-old child who was seen to swallow a penny. Aside from refusal to take all proffered food, there were no other symptoms to suggest the presence of a coin in the esophagus. In one case the initial symptoms were unknown.

In Table 4 are recorded the symptoms present at the time of examination of the patients from 2 to 24 hours after ingestion of the foreign object. All patients fell into this time interval with the exception of one patient seen three days after the accident and one in whom the time of the accident is unknown.

The character of the 38 foreign bodies lodged in the esophagus is as follows: coins, 14 (quarters, 6; pennies, 3; nickels, 1; half-dollar, 1; franc pieces, 2; dimes, 1); bones, 6; meat, 4; meat and bone, 1; thumb tacks, 2, and one case each of half of a shelled pecan, raw sweet potato, lead disk, button, crab shell, common straight pin, ear bob, collards, metal spring, metal ring, and part of a toy whistle.

There were 26 foreign bodies lodged in the cervical esophagus, 10 in the mid-thoracic esophagus, one in the lower thoracic esophagus, and in one instance the site of lodgment was not determined (Case 22). The site of foreign body arrest was determined by x-ray before esophagoscopy in the cases of opaque foreign bodies and when first visualized at esophagoscopy in the case of nonopaque foreign objects.

Twenty-eight patients in this series were referred by other physicians. Twenty of these patients were referred with a diagnosis made from a history of foreign body accident and initial symptoms. In 5 of these 20 cases x-rays were made by the referring physician but failed to reveal the presence of a foreign body. In four cases each the diagnosis was established by the referring physician on the basis of his x-ray or fluoroscopic examination. The prompt diagnosis in these cases redounded to the patient's benefit and facilitated my own efforts.

As a part of the treatment in this series of cases the foreign body was removed by esophagoscopy in 34 patients. In one case a large metal spring escaped into the stomach after the forceps' grasp was lost during the process of extraction. In one patient the foreign body was ejected by the patient after an unsuccessful esophagoscopy; one patient died during esophagoscopy because of acute cardiac arrest, and in the remaining case a straight pin in the esophagus was ejected into the nasopharynx and removed with forceps during the preliminary examination in the office.

All work was performed without general anesthesia. The usual preoperative medication for small children was atropine sulfate and for older children and adults morphine sulfate, atropine sulfate, and pentobarbital (Nembutal) in an appropriate dosage. Routine laboratory work in all hospitalized cases consisted of a complete blood cell count and urinalysis, with blood serologic studies in selected cases.

The average time required for removal of coins was three to five minutes. The longest time required to remove a foreign body was 30 minutes.

There are certain characteristic features of esophageal foreign bodies regarding their site of lodgment, character, and manner of lodgment.

The lodgment of foreign bodies in the esophagus is influenced by (1) the shape of the foreign object, (2) resiliency of the object, (3) size of the foreign body, (4) narrowing of the esophagus from any cause, and (5) paralysis of the normal esophageal propulsive mechanism.[94] Most foreign bodies are arrested in the cervical esophagus at the level of the superior aperture of the thorax. A physiologic narrowing is present at this point, produced in part by muscular contraction and mainly by the crowding of adjacent viscera into the fixed and narrow upper thoracic aperture. The chief factor, however, is the relative weakness of the propulsive mechanism as compared with the powerful inferior constrictor muscle. This muscle normally projects food into the cervical esophagus, where the weaker muscular wall of the esophagus by peristaltic wave carries the easily propelled bolus downward. In the

case of a foreign body that is hard and unmouldable, resistance to onward movement is encountered as compared with the smoothly sliding bolus, and the relatively weak peristaltic wave cannot overcome this resistance.[94] A lesser number of foreign bodies may lodge in the upper thoracic esophagus and an even smaller number in the lower thoracic esophagus.§

The character of most esophageal foreign bodies is well established. While coins and disks, bones, pins, and dentures may invade the larynx, trachea, and bronchi, they are seen more commonly in the esophagus. Nuts, watermelon seeds, vegetables, fruit pits, etc., on the other hand, rarely become impacted in the normal esophagus. Jackstones have been a fairly common esophageal foreign body, but up until 1937 they had not been reported as invading the lower respiratory tract.[117] Leeches are common laryngeal foreign bodies in some parts of the world and may be found occasionally in the esophagus.[70] Live fish also occasionally invade the esophagus in certain foreign countries.‖

The manner of lodgment of certain foreign objects in the esophagus is characteristic. Open safety pins usually lodge with point up and with the widest diameter of spread in the coronal plane. Occasionally they lodge point downward. In such an event it is interesting to conjecture how such an open safety pin impacts at such a low level as the cricoid narrowing without becoming embedded in the soft parts higher up. Fishman,[148] after reviewing the anatomy and physiology of swallowing, concluded as follows:

> (1) that objects of small mass, comparable to liquids, such as safety-pins, are not grasped by the pharyngeal constrictors, but are quickly and forcibly propelled through the mouth and mesopharynx; (2) that the hypopharynx, because of its attachments anteriorly, is open and ready to receive objects, rather than closed, permitting rapid descent into the postcricoid space.

Rarely safety pins may pass into the stomach point up and later be ejected back into the esophagus point down.¶

In like manner coins and disk-like objects enter the esophagus and lower air passages in a characteristic pattern. They enter the former with their long axis in the coronal plane and the latter with their long axis in the sagittal plane. The x-ray appearance is said to be pathognomonic of coin or disk in the esophagus or lower air passages in accordance with this position. Jackson and Jackson[94] discuss this problem as follows on page 33:

> Coins and other disklike objects naturally lie in the frontal plane, because the shape and action of the tongue anteriorly combine with those of the posterior pharyngeal wall posteriorly to make the long diameter of the pharyngeal lumen transverse. On the other hand, to enter the glottis, a flat object must lie in, or nearly in, the sagittal plane. In other words, the muscular activities of the pharynx throw flat foreign bodies into the frontal plane, whereas they must be in the sagittal plane to pass the glottis.

Again, on page 34:

> When flat objects like a coin go down, the cricoid cartilage lifts away from the spine, thus opening the pharynx sufficiently to allow the coin to pass with its greater diameter coronally placed. On the contrary, when such an object goes down through the larynx it takes a position

§ References 94, 97, 102, and 145.

‖ References 146 and 147.

¶ References 93, 149, and 150.

with its greater plane corresponding to the sagittal plane of the glottis. . . . The relative positions are important for differential diagnosis as to whether the air or the food passage is invaded.

Finally, on page 154:

It must be remembered that flat objects like coins always lie with their greatest diameter in the coronal plane when in the esophagus, in the sagittal plane when in the trachea.

Killian [82] stated that coins, pieces of bone, and artificial dentures are situated in a flat position in the esophagus in a frontal direction. Manges,[95] who did much to develop a roentgen technique and x-ray interpretation of foreign bodies in the air and food passages, stated that in the esophagus the coin always has its flat surface forward.

As with the safety pins in the esophagus, exceptions to the characteristic pattern of lodgment occur with coins in that they may lodge in the esophagus with the long axis in the sagittal plane instead of the customary coronal plane. Two such exceptions were noted in my practice in a relatively short period of time. In reviewing the literature on this subject, I read in detail 230 publications, excluding textbooks, concerned in the whole or in part with esophageal foreign bodies. The only counterpart to my own two cases found in the literature was reported by Schwartz [151] in 1947. Schwartz's patient was a 7-year-old child who swallowed a quarter that lodged in the esophagus in the sagittal plane. The position was demonstrated by x-ray, and the author offered as the only logical explanation of the roentgenograms the following:

The coin passed through the esophageal entrance in the usual manner, but somehow it made its way toward the extreme left of the esophagus and turned in an anteroposterior position, perhaps through striking the bulge of the arch of the aorta. Thus the posterior edge of the coin became wedged in an esophageal pocket beside the body of a vertebra, while the anterior edge was similarly wedged in a pocket to the left of the trachea. It is even probable that the downward escape of the quarter was prevented by the left main bronchus, which leaves the trachea between the fourth and fifth thoracic vertebrae. Thus the coin was caught laterally by the distended esophageal wall and the arch of the aorta, posteromedially by the vertebrae, anteromedially by the trachea and anteroinferiorly by the left main bronchus.

Esophagoscopy revealed precisely the condition described. Since the lumen of the trachea is smaller than the body of a thoracic vertebra, its lateral wall is nearer the midline than that of a vertebra. Furthermore, the aorta seems to press the trachea slightly to the right. This explains why the anterior edge of the quarter was found to stand nearer the midline than the posterior, so that the coin was not in perfect anteroposterior position but was tilted slightly, the anterior edge medialward.

In this position it could not be removed from the esophagus because of the tight upper entrance or slit in the coronal plane. The coin was grasped with the forceps, and after a little manipulation it was freed from its anterior pocket, after which extraction in the coronal plane became a simple matter.

As a result of his search of the literature and personal communications, Schwartz believed that this was the first case of its kind to be recorded. Jackson and Jackson do not allude to this position for coins in the esophagus in their older texts. In their latest book, "Bronchoesophagology," these authors make the following statement, "Coins and disks usually lie in the coronal plane; if not, they must be rotated into the coronal plane before they reach the cricoid level." They do not cite any specific examples of coins in the esophagus occupying the sagittal plane. Jesberg [152] alludes to a half-dollar in the dorsal esophagus as being held in the oblique position by the vertebral column. Withers [158] reported an unusual case in which a dime in the larynx

rested on the upper surface of the vocal cords, with lateral margins projecting into the ventricles. He stated that no similar case was found in the literature. Manges [95] stated that he had seen a few cases in which a coin-shaped foreign body was found in the trachea and disposed in the same manner as if it had been in the esophagus. These cases cited are the only ones I have been able to find recorded in the literature in which the typical pattern of lodgment of coins in the lower air passage and esophagus was altered or reversed.

The following case reports represent some unique feature of esophageal foreign bodies encountered during the period under discussion. The management of foreign bodies in the esophagus in a small hospital is sometimes a matter of expediency as well as principle.

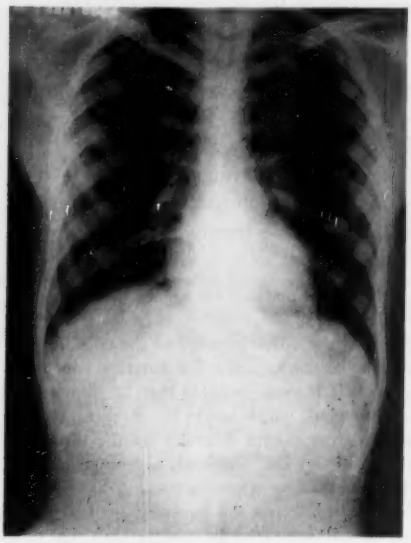


Fig. 1 (Case 13).—X-ray (anteroposterior view) showing an American half dollar occupying the sagittal plane in the thoracic esophagus.

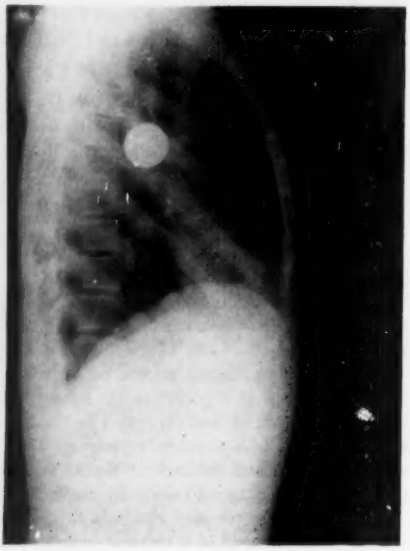


Fig. 2 (Case 13).—X-ray (lateral view) showing an American half dollar occupying the sagittal plane in the midthoracic esophagus.

REPORT OF CASES

Coins in the Esophagus Lodged in the Sagittal Plane

Case 13.—E. H., a white youth, aged 17 years, referred by Dr. F. Norris, was first observed July 2, 1948. The patient was spending his 17th birthday at the home of his cousin. About 3 o'clock in the afternoon the patient was attempting to amuse her by placing an American half dollar on his tongue and making it disappear into his mouth. This was accomplished by extruding the tongue, placing the coin on the dorsum of the tongue near the tip, curling the tip of the tongue over the coin, and withdrawing both into the oral cavity. Encouraged by his preliminary success in this maneuver and by the acclaim of his cousin, he repeated the usual maneuver in a more rapid and enthusiastic manner, with the result that the coin slipped off the tongue into the pharynx and was swallowed. The patient immediately developed a diffuse aching pain in the right anterior chest region, especially marked in the lower half. This was intermittent at first but rapidly became constant. A few minutes after the accident the patient induced vomiting once by digital manipulation in the pharynx. He was then taken by an aunt to the office of the referring

physician, who made a fluoroscopic examination and made a diagnosis of esophageal foreign body. He was given a small amount of water to drink in the physician's office, and this was promptly vomited. The patient was seen about one hour later. In the interim he had developed an additional severe aching pain in the region of the angle of scapula on the left side. There was no further vomiting and no further intake by mouth.

The general medical history was not important except that the patient was subject to attacks of bronchial asthma. Examination of the ears, nose, and throat and the general physical examination were not noteworthy. Anteroposterior and lateral views of the esophagus were reported by Dr. P. T. Myers as follows: "Examination of the chest shows the presence of a coin in the esophagus which has the size and appearance of a fifty-cent piece. It is situated at the level

Fig. 3 (Case 13).—American half dollar placed in the esophagus of a cadaver to illustrate its position in the sagittal plane to the left of the vertebral column.

of the sixth and seventh dorsal vertebrae. The thin edge of the coin is seen in the AP view and the large mass of the coin is seen on the lateral view. There are no lung changes." (Figs. 1 and 2).

The patient was hospitalized and esophagoscopy performed late in the same afternoon, with some thought being given before hand to the unique position and possible method of extraction. An 8-mm. esophagoscope was introduced into the esophagus after preliminary anesthetization of the pharynx with 2% tetracaine (Pontocaine) hydrochloride solution. The foreign body was located at the midthoracic esophagus, just above the narrowing produced by the arch of the aorta. It was in the anteroposterior plane and situated in a pocket to the left of the vertebral column (Fig. 3). While an appraisal was being made of the position, there was a sudden ejection of stomach contents, which required several minutes of aspiration for clearance. When the coin was again inspected, it was seen that the anterior edge was inclined slightly toward the midline. The position at this point was essentially that of the coin in the case reported by Schwartz.[151] Since the anterior edge of the coin had been rotated slightly by the vomiting, it was felt that

manipulation by forceps into the coronal plane could best be accomplished by rotating the anterior edge of the coin, since this apparently represented the line of least resistance. Forward-grasping forceps were accordingly applied to this area of the coin. The forceps were rotated so that the blades moved in a lateral direction instead of the usual anteroposterior manner. After the forceps were placed on the flat surface of the coin near the anterior edge, a twisting motion was imparted to the forceps in a clockwise manner, the handle of the forceps moving from right to left. Rotation was gradual after several such maneuvers, and the coin was eventually rotated to the coronal plane. Extraction from this position was easy. The coin, however, struck the edge of the metal bite block as it was being withdrawn from the mouth and was dislodged from the grasp of the forceps, being swallowed a second time. It was located again in the mid-esophagus in the coronal plane, and further extraction was uneventful. Relief from pain was immediate, the further postoperative course was uneventful, and the patient was discharged from the hospital the next day.

Case 14.—J. B. H., a white man, aged 65 years, referred by Dr. Sabiston, was first observed Jan. 25, 1949. The patient, being devoid of funds, borrowed a quarter from a friend with which

Fig. 4 (Case 14).—X-ray (anteroposterior view) showing an American quarter in the thoracic esophagus in the sagittal plane.

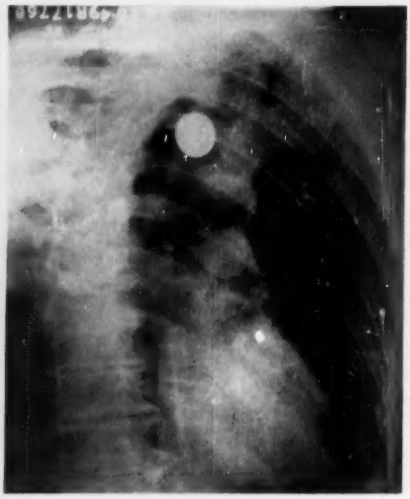


Fig. 5 (Case 14).—X-ray (oblique view) showing an American quarter in the esophagus in the sagittal plane.

to purchase breakfast. The money was diverted from this purpose, however, since shortly afterward the patient was "cuttin' the fool with a young girl" by hiding the coin in his mouth. While performing the "hidden coin" trick, he accidentally swallowed the quarter. The accident was accompanied by dry cough and discomfort at the sternal notch area. He attempted to induce vomiting by finger manipulation in the pharynx. He then drank some liquids but strangled in the attempt. The referring physician was consulted, and he made a diagnosis of esophageal foreign body on the basis of history and symptoms and referred the patient for treatment.

Examination of the ears, nose, and throat and the general physical examination were not noteworthy. Anteroposterior and lateral films of the esophagus were reported by Dr. P. T. Myers as showing the presence of a metallic foreign body in the esophagus, occupying the sagittal plane at the level of the sixth and seventh dorsal vertebrae (Figs. 4 and 5).

The patient was not hospitalized because no beds were available at the time. Esophagoscopy was performed as an outpatient after preliminary anesthetization of the pharynx with 2% tetracaine solution. An 8-mm. esophagoscope was introduced without difficulty to the proximity of the coin. It was an American quarter lodged in the sagittal plane in the midline of the esophagus,

just above the aortic crossing (Fig. 6). Marked pulsation was transmitted to the foreign body from the aorta. As a preliminary measure as much traction as was consistent with safety was made on the coin to see if any motion could be imparted to it while in the sagittal plane. The coin was firmly fixed in this position and could not be moved. Forward-grasping forceps were then applied to the flat surface in the same manner as in the preceding case, except that application was made near the posterior edge. A twisting motion was then applied to the forceps in a clockwise manner, the posterior edge of the coin being dislodged to the left after several such maneuvers. The coin was thus rotated to the coronal plane, and further extraction was uneventful. The patient was observed daily in the office for several days, and the further course was uneventful.

Fig. 6 (Case 14).—American quarter placed in the esophagus of a cadaver to illustrate its position in the sagittal plane. In the patient the coin was nearer the midline of the vertebral column than is shown in the photograph.

Comment.—There are several unique features about these two cases, the most striking, of course, being their lodgment in the sagittal plane. No explanation can be offered as to how this happened. In both instances the patients were older than the children usually seen with coins in the esophagus. In like manner the coins were lodged in the midesophagus rather than the usual cervical region. American half-dollars and even silver dollars have been reported as esophageal foreign bodies in young and older adults, but these cases are not common. Prompt diagnosis in both instances very probably prevented serious complications, since it is difficult to see how these foreign bodies could have remained in situ very long without producing pressure necrosis of the esophageal wall. The first of these two cases is the first esophageal foreign body that I removed in this community. Both cases were seen

within a relatively short time of each other. It is suggested that the etiologic synergy of these two cases might be carelessness and a variant of Jackson and Jackson's usual classification of "emotions."

Serious Hemorrhage from the Esophagus as a Result of Injury to an Intramural Vessel

CASE 15.—O. L. S., a white man, aged 62 years, referred by Drs. F. and C. Wooten, was first observed July 14, 1951. About two hours earlier the patient was at his summer camp some 40 miles distant. After the first swallow of a crab stew prepared for supper he experienced a severe cutting pain in his chest and spat up some bright red blood. He then ate some bread, which was promptly regurgitated, but was able to swallow some lemon juice. After removing his artificial dentures, the patient then made vigorous efforts with finger manipulation in the pharynx in an unsuccessful effort to induce vomiting. The patient made the trip from camp to home in severe pain, which was obvious enough when he was seen in the office with the referring physicians.

Examination of the ears, nose, and throat was not noteworthy except for the presence of bloody secretions in the pyriform sinuses and numerous petechial hemorrhages of the soft palate, tonsillar pillars, pharynx, and lateral pharyngeal bands of lymphoid tissue, due to trauma from previous digital efforts to induce vomiting. The general physical examination by Dr. C. Wooten was reported to be essentially normal. X-rays of the esophagus failed to reveal the presence of a foreign body.

The patient was hospitalized, and a 7-mm. esophagoscope was introduced after preliminary anesthetization of the pharynx with 2% tetracaine solution. After bloody secretions and food particles were removed from the upper esophagus by suction, a foreign body consistent in appearance with some portion of a crab shell was noted about 2 in. (5 cm.) below the cricopharyngeus muscle. The object completely filled the lumen of the esophagus, the presenting portion appearing to be rather thin and flexible. The lateral margins could not be visualized well, even after displacing the lateral walls of the esophagus with the tip of the esophagoscope. Because of the pain and bleeding it was felt that a sharp edge was present at some point on the foreign body. The object was grasped with forward-grasping forceps and traction made. The foreign body moved out of the esophagus about 1 in. (3 cm.) and then impacted firmly. It was felt by the sensation imparted to the forceps that the impaction was in the right lateral wall of the esophagus. The object was then pushed down about an inch, and an attempt was made to rotate the right lateral margin toward the midline. Because of the flexibility, however, this effort was not successful. The object was then moved out by traction approximately 1 in. At this point impaction again occurred, and simultaneously with this arrest of progress there was a sudden and profuse hemorrhage through the lumen of the esophagoscope, the blood pouring onto the floor in a rapidly widening pool. The forceps were then removed and the esophagoscope rotated so as to bring the tip up against the right lateral wall of the esophagus at the point where the bleeding was suspected of originating. With the use of the thumb of the left hand as a fulcrum, as much pressure as was consistent with safety was made on the right lateral wall of the esophagus with the esophagoscope. The bleeding stopped immediately after this maneuver, but the pressure was maintained for five minutes by the clock. At the end of this time the esophagoscope was withdrawn. There was no recurrence of the bleeding at any time.

It was difficult to estimate the amount of the blood loss at the time, but the patient was now in profound shock. Pulse and blood pressure could not be obtained. The patient was cyanotic, dyspneic, sweating, and cold. He was returned to his room and adequate measures to combat shock instituted by Dr. C. Wooten. Complete recovery from surgical shock was slow, requiring several days.

There was a moderate elevation of temperature for five days after esophagoscopy, and the patient continued to gag and vomit at intervals. After a suitable interval another esophagoscopy was contemplated with the intention of cutting the foreign body with shears in order to facilitate removal. However, on the night of July 18, 1951, the patient suddenly ejected what proved to be a portion of a crab claw very much curled upon itself. Among the margins of the claw were

projecting spines with needle-sharp points (Fig. 7). The further progress was uneventful after spontaneous expulsion of the foreign body, and the patient was discharged from the hospital July 26, 1951. There were no untoward sequelae.

Comment.—While bleeding from the esophagus may hinder attempts to remove foreign bodies or even occasionally be of such a degree as to cause temporary postponement of the attempts, hemorrhage of such a degree, as was manifested by this patient, seems to be quite rare, if one may judge from the literature. The cause of the hemorrhage in this particular case was unquestionably due to injury to an intramural esophageal vessel by one of the projecting spines along the lateral margin of the claw.

Bone in the Esophagus: Difficult Localization and Extraction; Peculiar Pain Reference

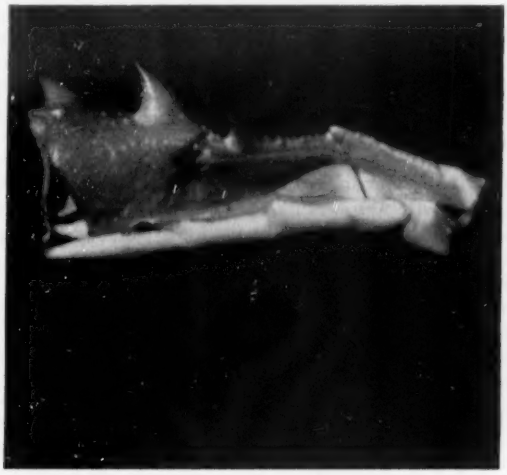


Fig. 7 (Case 15).—Crab claw with projecting spines causing serious hemorrhage from injury to an intramural esophageal vessel.

Case 16.—J. W. S., a white woman, aged 30 years, referred by Dr. F. Sabiston, was first observed May 8, 1950. A few hours before, the patient was eating cabbage which had been seasoned with ham hock. As was her usual custom when eating cabbage or other types of leafy vegetables, she filled her mouth and swallowed, thereby foregoing the customary preliminary process of mastication. Immediately after swallowing she felt something stop in her food passage and experienced a sharp pain which she localized at the xiphoid process. Finger manipulation in the pharynx failed to induce vomiting. She then ate some corn bread, which passed into the stomach but occasioned considerable pain. The referring physician was consulted, who made a diagnosis of esophageal foreign body on the basis of history and symptoms.

Examination of the ears, nose, and throat and the general physical examination were not noteworthy. Anteroposterior and lateral films of the esophagus failed to show any evidence of a foreign body. The use of chloriodized oil (Iodochloral) also failed to outline any definite esophageal foreign object. Because the patient was able to swallow liquids and soft food, in view of the definite point localization of pain at the xiphoid process, and in the absence of positive x-ray findings, it was felt that the foreign body was lodged in the terminal esophagus.

The patient was hospitalized for esophagoscopy. After preliminary anesthetization of the pharynx with a 2% tetracaine solution, a 7-mm. esophagoscope was introduced throughout the

length of the esophagus, without discovery of a foreign body. Esophagoscopy was performed a second time, with negative results, and the patient was then returned to her room. It was felt that the foreign body, thought to be in the terminal esophagus, had slipped into the stomach as the esophagoscope approached the first time. Two days later the patient was able to take soft foods with very little discomfort, and she was discharged from the hospital for continued observation in the office.

The patient returned for observation May 15, 1950. She stated that on the day following discharge from the hospital she became unable to swallow soft food but continued to take liquids without difficulty. At the same time the sharp pain previously localized to the xiphoid process now radiated into the left chest anteriorly, left shoulder, and down to the left elbow. On May 15, 1950, she had vomited intermittently all day.

The patient was hospitalized, and x-rays again were negative. On May 16, 1950, after the usual preliminary anesthetization of the pharynx with a 2% solution of tetracaine, the upper esophagus was explored by means of an esophageal spectulum. No foreign body was found. A 7-mm. esophagoscope was introduced throughout the length of the esophagus twice, with negative results. It was now felt that the most likely site for the foreign body was just below the cricopharyngeus muscle, even though this area had been carefully explored. With this in mind, the esophagoscope was introduced to the cricopharyngeus muscle in the usual manner. It was then gently rotated so as to bring the tip posteriorly, with the beveled portion pointing anteriorly in contrast to the usual position. The tip of the esophagoscope was advanced slowly, with some pressure being exerted to displace the soft tissues posteriorly. This maneuver was successful, since the tip of the advancing esophagoscope brought to view the edge of a large thin flat bone lodged just below the cricopharyngeus muscle. Inserting the tip of the esophagoscope posterior to the bone the presenting edge was maneuvered into full view in the middle of the esophageal lumen. The esophagoscope was then rotated to the usual position, and a good purchase was easily secured on the bone. Considerable traction, however, was necessary for its extraction. There was surprisingly little edema of the mucosa of the esophagus at the site of the foreign object. The patient was returned in good condition to her room. There was immediate relief from the bizarre pain. She was discharged from the hospital May 18, 1950, free of symptoms. The foreign body was a large flat thin bone which measured 3 by 2.5 cm., and there were no sequelae incident to its sojourn or removal.

Comment.—The etiologic synergy in this case were carelessness in the preparation of food and the failure to masticate. Any person with a full set of teeth could not possibly have failed to detect the presence of such a large foreign object in the food if it had been even partially masticated. The localization of pain at the xiphoid process was certainly misleading throughout the entire period of management of this patient. Introduction of the esophagoscope into the esophagus in the reverse of the usual position was undertaken carefully and with full recognition of the possibilities regarding perforation. It was undertaken in an attempt to solve a problem in foreign body localization that had rapidly become exasperating. The time required for removal from start to finish was 30 minutes, which was the longest interval required to remove any foreign body in this series of patients.

Coin in the Esophagus, Resulting in Death from Acute Cardiac Arrest During Attempts at Extraction

CASE 17.—F. H. Jr., a Negro boy, aged 2 years, was first observed Dec. 5, 1949. Three days previously, while playing alone, the patient swallowed a penny. He informed his mother of this fact shortly after the accident. For three days the patient swallowed liquids but vomited solids. When asked where his money was, the patient invariably opened his mouth and pointed to his throat. There were no other symptoms.

Examination of the ears, nose, and throat and the general physical examination were not noteworthy. X-rays revealed the presence of a metallic foreign body in the cervical esophagus at the level of the thoracic inlet.

The patient was hospitalized for esophagoscopy. A 5-mm. esophagoscope was introduced without preliminary anesthetization of the pharynx. The penny was first noted at the middle third of the esophagus but passed to the lower third before a purchase was secured. Extraction was started, but the forceps slipped off several times at approximately the middle third of the esophagus. A 6-mm. esophagoscope was then introduced and a larger forward-grasping forceps used to secure a purchase. The purchase was again lost at approximately the same area. As the esophagoscope was again approaching the area of the aortic crossing, the pulsation normally noted in this area suddenly ceased. Simultaneous with this observation on my part, the head holder announced that respiration, previously normal, had suddenly stopped. The esophagoscope was withdrawn at once, a bronchoscope introduced, and oxygen blown in through the bronchoscope. Respiration returned immediately. During this time Dr. W. E. Keiter, who was immediately available, gave intracardiac epinephrine while respiratory and cardiac stimulants were being administered hypodermically by the nursing personnel. The cardiac function was restored, but the rhythm remained irregular. The patient finally died about two hours later.

Comment.—It is believed that the cause of death in this patient was acute cardiac arrest rather than tracheal compression. A similar type case would best be treated by thoracotomy and cardiac massage in the present era. No reason can be assigned for the difficulty in extracting the coin. The region in which the purchase was repeatedly lost corresponded to that of the aortic crossing. Presumably the repeated manipulation in this region set up the reflex mechanism responsible for the sudden arrest of cardiac and respiratory function. The 6-mm. esophagoscope should not have been too large for a child of this age group. This is the only fatality in this series of cases.

Atelectasis Following Removal of a Cartilaginous Esophageal Foreign Body

CASE 18.—O. M., a white woman, aged 46 years, referred by Dr. F. Norris, was first observed Dec. 30, 1952. A few hours previously the patient swallowed a piece of gristle while eating hog meat. She stated that it felt as if the gristle was lodged crosswise in her throat. She took solid and liquid foods, which were promptly regurgitated. Finger manipulation in the pharynx was followed by vomiting, the contents showing some blood streaking. No further food or digital manipulation was resorted to. The patient consulted the referring physician, whose fluoroscopic studies gave negative results. She was referred with a diagnosis of an esophageal foreign body on the basis of history and symptoms.

Examination of the ears, nose, and throat and the general physical examination were essentially normal. Routine x-rays of the esophagus gave negative results. The use of chloriodized oil, however, revealed the outline of a large foreign object in the upper thoracic esophagus.

The patient was hospitalized for esophagoscopy. After the usual preliminary anesthetization of the pharynx with a 2% tetracaine solution, a 7-mm. esophagoscope was introduced to the level of the foreign body. It proved to be a large mass of cartilaginous tissue which was extremely friable, requiring introduction of the esophagoscope four times in order to obtain complete removal of all of the tissue. The patient was extremely cooperative, and the entire procedure, which lasted 25 minutes, was entirely uneventful.

Two hours after esophagoscopy the patient experienced a sharp pain in the chest and between the shoulders, associated with mild difficulty in breathing. X-rays interpreted by Dr. W. G. Budington and clinical examination of the chest by Dr. L. E. Williams established a diagnosis of atelectasis of the right lower lobe of the lung. Bronchoscopy revealed an essentially normal tracheobronchial tree. The further course was uneventful, and the patient was discharged from the hospital Jan. 20, 1953. There were no sequelae.

Comment.—Atelectasis following removal of an esophageal foreign body apparently is not common. It was considered that a fragment of the cartilage may have invaded the tracheobronchial tree during the process of removal from the esophagus, but such was not the case. The cause of the atelectasis is unknown.

Nuts and Vegetables Causing Esophageal Obstruction in Children

CASE 19.—C. R. W., a white boy, aged 18 months, referred by Dr. R. J. Walker, was first observed Nov. 29, 1948. A few hours before, the patient was eating pecans, which were being fed to him by his mother. He suddenly grabbed some of the nuts from her hand and, putting them in his mouth, started to run. However, he fell in the attempt. There was a brief spell of coughing, which subsided without recurrence. This was followed by gagging and drooling, which persisted. The patient was given liquids, which he swallowed, but he regurgitated soft foods. The parents observed that when he swallowed liquids he "sounded hollow." The referring physician made a diagnosis of esophageal foreign body on the basis of history and symptoms.

Examination of the ears, nose, and throat and the general physical examination were essentially normal. Routine x-ray studies gave negative results.

The patient was hospitalized for esophagoscopy. No anesthesia was used, and a 4-mm. esophagoscope was introduced. The foreign body was the large half of a shelled pecan which had lodged transversely just below the cricopharyngeus muscle. It fragmented on removal with forward-grasping forceps. The further course was uneventful, and the patient was discharged from the hospital the following day.

CASE 20.—L. C. Jr., a white boy, aged 16 months, referred by Dr. D. H. Jones Jr., was first observed Feb. 11, 1950. A few hours earlier, while eating a raw sweet potato, the patient suddenly had a choking and coughing spell and began to spit out fragments of the potato. Since that time he had vomited and coughed intermittently, drooled constantly, refused all food and liquids, and became completely quiet and inactive. The patient was referred with a diagnosis of esophageal foreign body on the basis of history and symptoms.

Examination of the ears, nose, and throat and the general physical examination gave normal findings. Routine x-rays failed to reveal the presence of a foreign body.

The patient was hospitalized for esophagoscopy. A 5-mm. esophagoscope was introduced without preliminary anesthetization. The foreign body was a large wedge-shaped piece of raw sweet potato, which completely filled the lumen of the esophagus just below the cricopharyngeus muscle. It was removed without incident, and the patient was discharged from the hospital the following day.

CASE 21.—S. G. D., a white girl, aged 3 years, referred by Dr. W. E. Keiter, was first observed Aug. 15, 1952. The patient was brought in by the mother, who had a fixed idea that her child had swallowed glass a few hours before, although she had no idea as to how this had happened. The patient had been left alone with an older sister, who conveyed the information that the patient had swallowed the glass. The symptoms developed at noon, when the sister attempted to feed the patient, who refused to eat or drink. A few hours later she began to cough and drool. When the mother returned and was informed of the supposed accident, she turned the patient upside down and struck her finger in her mouth in a vain attempt to produce either vomiting or the foreign body. Since that time a croupy cough had been present. The referring physician made a diagnosis of esophageal foreign body on the basis of history and symptoms.

Examination of the ears, nose, and throat and the general physical examination were essentially normal. The patient drooled constantly and was unable to swallow water. Routine x-rays failed to reveal the presence of a foreign body.

The patient was hospitalized, and a 5-mm. esophagoscope was introduced without preliminary anesthetization. The foreign body was encountered in the cervical esophagus at the region of the thoracic inlet. The appearance through the esophagoscope was confusing. The first foreign object removed proved to be a mass of tomato skins with some granular material intermixed. The greater mass, however, proved to be the large heavy stems of collards mixed with the leafy portion of the vegetable (collards are similar to cabbage and are common in eastern North Carolina). The amount of the foreign body removed was amazing. The child drank water freely after esophagoscopy and was discharged from the hospital the following day. It developed that the patient had eaten the collards the afternoon before the day of admission to the hospital. The following morning she ate a small amount of cooked tomatoes and grits. Symptoms developed at noon.

Comment.—Complete esophageal obstruction due to food is relatively uncommon in children, and shelled nuts as esophageal foreign bodies are rare. The causation in Case 21 was obviously improper preparation of food.

Foreign Body in the Esophagus Ejected into the Nasopharynx by Vomiting

CASE 22.—I. J., a Negro boy, aged 12 years, was first observed Jan. 25, 1952. The patient stated that a few hours earlier he was picking his teeth with a straight pin at school. The pin slipped from his hand and was swallowed. He experienced discomfort in the laryngeal region of the neck. There were no other symptoms.

It is my policy to examine the nasopharynx and laryngopharynx three separate times before giving an opinion as to the presence or absence of a foreign body. On this occasion the third such examination had been completed and no foreign body noted. The patient then vomited, and when the pharynx was again inspected, a common straight pin was seen in clear view embedded obliquely in the right lateral pharyngeal band of lymphoid tissue. It was easily removed with forceps.

Comment.—The pin unquestionably originally lodged in the esophagus and was ejected into the nasopharynx by vomiting.

CONCLUSIONS

As a result of this study of all proved exogenous foreign bodies of the external auditory canal, nasal cavity, and esophagus seen in office practice in this small community from Feb. 1, 1948, through July 31, 1953, the following conclusions can be drawn:

1. Foreign bodies of the external auditory canal, nasal cavity, and esophagus are not common.

2. Most foreign bodies of the external aural canal and nasal cavity produce few, if any, immediate symptoms, are usually seen promptly, and are usually easily removed. The majority of foreign objects in these regions, together with esophageal foreign bodies, may be removed without general anesthesia.

3. Unskilled attempts at removal of foreign bodies from the external auditory canal may produce more trauma to the ear than that wrought by the foreign object per se.

4. Sulfonamides administered orally and penicillin orally or by injection seem to help in preventing secondary infection in those ear cases with traumatic injury secondary to the foreign body or to attempts at removal.

5. Foul odor of undetermined origin may be the initial symptom of nasal foreign bodies in children.

6. Coins rarely lodge in the esophagus in the sagittal plane, and they cannot be removed from the esophagus with safety while in this plane. Rotation into the coronal plane may be accomplished with forward-grasping forceps applied to the flat surface of the coin near the anterior or posterior edge in accordance with the position of the coin. In the case of a coin lodged in a pocket to the left of the vertebral column above the aortic crossing, application of forceps may be made on the flat surface near the anterior edge. With the utilization of a twisting motion, the anterior edge of the coin may be rotated to the right in a clockwise manner. This seems to represent a line of least resistance for a coin occupying this position.

7. Serious hemorrhage from rupture of an intramural esophageal vessel may attend attempts at removal of an esophageal foreign body with lacerating potentialities.

8. Pain referral from an esophageal foreign body may be bizarre and misleading.

9. Complications of esophagoscopy for foreign body removal may include acute cardiac arrest with fatal termination and atelectasis.

10. Nuts and vegetables may cause esophageal obstruction in children.

Kinston Clinic.

REFERENCES

1. Bezold, F., and Siebenmann, F.: Testbook of Otology, Chicago, E. H. Colegrove Co., 1908, p. 104.

2. Griffin, E. H.: Foreign Bodies in the Ear and Their Removal, M. Rec. & Ann. **145**:409-410 (May 19) 1937.

3. Keen, J. A.: Foreign Bodies in the Ears and Nose, J. Laryng. & Otol. **45**:333-343 (May) 1930.

4. Hung, W. C., and Lin, P. J.: Foreign Bodies in Air and Food Passages: A Clinical Observation in Taiwan, A. M. A. Arch. Otolaryng. **57**:603-612 (June) 1953.

5. Mosher, W. F.: Foreign Bodies of External Canal, Middle Ear, and Mastoid and Their Complications, Arch. Otolaryng. **36**:679-686 (Nov.) 1942.

6. Lion, H.: Burn of the Tympanic Membrane in Welder, Arch. Ohren-Nasen-u. Kehlkopfh. **122**:195-197 (Aug.) 1929.

7. Heermann, J.: Cases of Burns of the Auditory Canal, Middle Ear and Labyrinth Occurring in Welders and Foundry Workers, Ztschr. Hals- Nasen- u. Ohrenh. **39**:546-556 (July) 1936.

8. Britton, J. A., and Walsh, E. L.: Health Hazards of Electric and Gas Welding, J. Indust. Hyg. & Toxicol. **22**:125-151 (April) 1940.

9. McNaught, R. C.: Foreign Body in the Eustachian Tube: Report of a Case, Laryngoscope **58**:67-73 (Jan.) 1953.

10. Simonton, K. M.: Foreign Bodies of the External Auditory Canal Causing Otitis Media, Arch. Otolaryng. **49**:16-19 (Jan.) 1949.

11. Taylor, H. M.: Screwworm (Cochliomyia Americana) Infestation in Man, Ann. Otol. Rhin. & Laryng. **59**:531-540 (June) 1950.

12. Baker, M. C.: "Green-Bottle Fly Worm" Infestations of the Ear: Myiasis of the Ear Caused by Phaenicia Sericata, Laryngoscope **63**:545-548 (June) 1953.

13. Markoff, K. K.: Worm in the Ear Drum, J. A. M. A. **83**:918 (Sept. 20) 1924.

14. Seshachalam, T.: An Unusual Foreign Body in the Ear, Indian M. Gaz. **62**:145 (March) 1927.

15. Foster, B.: A Tick in the Auditory Meatus, M. J. Australia **1**:15-16 (Jan. 3) 1931.

16. Zappala, A.: Emofil nel condotto uditivo esterno, Arch. ital. otol. **61**:278-280 (May-June) 1950.

17. Piquet, J., and Decroix, G.: L'otite moyenne chronique suppurée pars corps étrangers du conduit auditif et de la trompe d'eustache, Rev. laryng. **71**:136-145 (March-April) 1950.

18. Ochs, I. L.: Foreign Body of the Middle Ear, Arch. Otolaryng. **47**:670-672 (May) 1948.

19. Hempstead, B. E.: Bullets in the Ear: Report of 3 Cases, J. A. M. A. **98**:2281-2282 (June 25) 1932.

20. Warren, W. C., Jr., and Bivins, T. E.: Unusual Otitic Foreign Bodies: Report of 2 Cases, Arch. Otolaryng. **49**:196-200 (Feb.) 1949.

21. Proctor, B.: Removal of Impacted Foreign Bodies from the External Auditory Canal by the Endaural Approach, Arch. Otolaryng. **36**:131-133 (July) 1942.

22. Bettington, R. H.: Peculiar Sequel to a Foreign Body in the Ear, M. J. Australia **1**:535-536 (March 19) 1938.

23. Longaker, E. P.: Two Interesting Foreign Body Cases of the Mastoid and the Nose, Ann. Otol. Rhin. & Laryng. **38**:1158-1162 (Dec.) 1929.

24. Wood, V. V.: Scissors Point Embedded in the Inner Tympanic Wall, with Facial Paralysis, Ann. Otol. Rhin. & Laryng. **39**:111-114 (March) 1930.

25. Politzer, A.: Text-Book of Diseases of the Ear, Ed. 6, Philadelphia, Lea & Febiger, 1926, p. 241.

26. Toynbee, J.: Diseases of the Ear, Philadelphia, Blanchard & Lee, 1860, p. 71.

27. Cruz, B. C.: Cuerpos estranos en los oidos y fosas nasales en la infancia, Rev. cubana pediat. **19**:296-312 (May) 1947.

28. Wells, C. J. L.: Foreign Body Removed from the Ear After 33 Years, Brit. M. J. **2**:637-638 (Oct. 8) 1927.

29. Achaval, A.: Cuerpos extranos del conducto auditivo externo, Semana méd. **1**:117-122 (Jan. 17) 1946.

30. Tapia, C. A.: Los cuerpos extranos del conducto auditivo, An. Soc. mex. oftal. y oto-rino-laring. **15**:101-109 (March-April) 1940.

31. Hilton, G. E. M.: A Foreign Body in the External Auditory Canal, Canad. M. A. J. **33**:308 (Sept.) 1935.

32. Roberts, W. H.: A Foreign Body in the External Auditory Canal Resembling a Sequestrum, Ann. Otol. Rhin. & Laryng. **11**:530-531 (Aug.) 1902.

33. Lowry, E.: Foreign Body Removed from the Ear, Brit. M. J. **2**:875 (Nov. 12) 1927.

34. Chavanne, L.: Vertiges et cephalée par corps étranger de l'oreille y sejournant depuis 20 ans, Oto-rhino-laryng. internat. **19**:69 (Feb.) 1935.

35. Abdulla, M.: Foreign Body in the Ear a Cause of Persistent Hiccough, Indian M. Gaz. **65**:445-446 (Aug.) 1930.

36. Kennedy, W. P.: Foreign Body in the Ear for 36 Years, Brit. M. J. **1**:14 (Jan. 7) 1928.

37. Chalier, J., and Rousset, J.: Foreign Body in the External Auditory Canal Causing Tetanus in a Child 3 Years Old, Lyon med. **141**:675 (June 10) 1928.

38. Machado, R.: Tetanus cefalico consecutivo a cuerpo extrano en el oido derecho, Vida nueva **40**:94-97 (Aug. 15) 1937.

39. Hara, H. J.: Foreign Body in the External Auditory Canal Trapped by Exostosis, Laryngoscope **47**:126-132 (Feb.) 1937.

40. dos Reis, G.: Um caso interessante de corpo estranho do ouvido, Rev. brasil. oto-rino-laring. **8**:341-344 (Sept.-Oct.) 1940.

41. Dixon, O. J.: A New Method for the Removal of Foreign Bodies in the External Auditory Canal, J. A. M. A. **83**:357 (Aug. 2) 1924.

42. Richards, G. L.: Foreign Bodies in the Ear and Nose, Am. J. Surg. **6**:514-516 (April) 1929.

43. von Troltsch, A.: Diseases of the Ear, New York, William Wood & Company, 1864, p. 48.

44. Rosenwasser, H.: Foreign Bodies in the External Auditory Canal, Am. J. Surg. **36**:96-101 (April) 1937.

45. Wolfe, J. J.: Unusual Foreign Body in the Nose, Arch. Otolaryng. **26**:736-737 (Dec.) 1937.

46. Trexler, C. W.: Nasal Foreign Bodies: Report of a Case Having Lodgment for 7 Years, Ann. Otol. Rhin. & Laryng. **43**:620-623 (June) 1934.

47. Kelemen, G.: Spontaneous Expulsion of a Foreign Body by Transmigration Through the Nasal Wall After 28 Years, Laryngoscope **55**:375-379 (July) 1945.

48. Cantoni, A. J.: Wooden Foreign Body in the Ethmoid Capsule, Ann. Otol. Rhin. & Laryng. **56**:953-956 (Dec.) 1947.

49. Howard, J. C., Jr.: Foreign Bodies of Maxillary Sinus, Ann. Otol. Rhin. & Laryng. **54**:186-191 (March) 1945.

50. Voorhees, I. W.: Unique Foreign Body in the Nose, J. A. M. A. **74**:672 (March 6) 1920.

51. Vadala, A. J., and Somers, K.: Knife Blade Traversing Maxillary Antrum and Remaining in Nose 6 Years, Mil. Surgeon **103**:207-210 (Sept.) 1948.

52. Dowling, J. I.: Accident—Lost End of a Sphenoid Probe Recovered from Nasal Meatus, Laryngoscope **43**:911-917 (Nov.) 1933.

53. Myerson, M. C.: Foreign Body in the Nose, Diagnosed as Carcinoma, Unexpectedly Removed, Laryngoscope **38**:390-392 (June) 1928.

54. Pearlman, S. J., and Pilot, I.: Foreign Body Granuloma of the Nose, Laryngoscope **33**:854-856 (Nov.) 1923.

55. Santos, B. P.: Abcesso da orbita por corpo estranho nasal, Arq. brasil. oftal. **2**:230-232 (March) 1939.

56. Camerer, C. B.: Foreign Body in the Nares, U. S. Nav. M. Bull. **19**:168-169 (Aug.) 1923.

57. Altounyan, E. H. R.: Foreign Body Removed After 10 Years, Lancet **2**:119 (July 16) 1927.

58. Marks, M. I.: Foreign Body (Twig) in the Nose, Ann. Otol. Rhin. & Laryng. **58**:289-292 (March) 1949.

59. Donnelly, J. C.: Nasal Foreign Bodies, Arch. Otolaryng. **34**:349-353 (Aug.) 1941.

60. Ts'en, S. P.: Two Unusual Nasal Cases in Huchow, Chinese M. J. **44**:546-551 (June) 1930.

61. Magee, F. E.: Foreign Body in the Nasal Chamber, Atlantic M. J. **29**:460 (April) 1926.

62. Poe, D. L.: Foreign Bodies in Children's Noses and Ears, Arch. Pediat. **54**:434-440 (July) 1937.

63. Steele, J. S.: A Case of Epileptiform Convulsions Caused by Shoe Button in the Nose, Laryngoscope **11**:288-289 (Oct.) 1901.

64. Brown, E. H.: Screwworm Infestation in the Nasal Passages and Paranasal Sinuses, Laryngoscope **55**:371-374 (June) 1945.

65. Wilson, J. G.: A Centipede in the Nose, Brit. M. J. **1**:446 (March 9) 1929.

66. Astavans, H. C.: Invasion of the Nasal Cavity by a Leech, Indian M. Gaz. **66**:678 (Dec.) 1931.

67. Khan, M.: Invasion of the Nasal Cavity by a Leech, Indian M. Gaz. **67**:389 (July) 1932.

68. Narasimhaiah, H.: A Leech in the Nasal Cavity, Indian M. Gaz. **67**:572 (Oct.) 1932.

69. Narayanan, K.: A Leech in the Nose, Indian M. Gaz. **71**:530 (Sept.) 1936.

70. Meyer, M.: Blutegel, die weitaus häufigsten Fremdkörper der oberen Luft- und Speisewege in Anatolien, Pract. oto-rhino-laryng. **4**:142-156, 1942.

71. Muller, F.: Sobre alguns corpos estranhos mais raros do nariz, Rev. brasil. oto-rhino-laring. **7**:287-288 (May-June) 1939.

72. Rao, S. S.: Foreign Body in the Maxillary Antrum, Brit. M. J. **1**:546-547 (March 31) 1928.

73. Koepcke, G. M.: Cherry Stone in the Maxillary Sinus, Minnesota Med. **17**:258 (May) 1934.

74. Brown, J. J. M.: Tetanus Following Impaction of a Foreign Body in the Nose, Lancet **2**:677-678 (Sept. 24) 1932.

75. Burton, A. H. G., and Balmain, A. R.: Foreign Bodies and Nasal Carriers of Diphtheria, Lancet **2**:977-978 (Nov. 9) 1929.

76. Canfield, N.: Foreign Bodies in the Food and Air Passages: Résumé of 62 Cases Including 1 of Special Interest, Connecticut M. J. **1**:239-242 (May) 1937.

77. Polson, C. J.: On Rhinoliths, J. Laryng. & Otol. **58**:79-116 (March) 1943.

78. Myerson, M. C.: Rhinolith Requiring External Operation for Its Removal, Laryngoscope **38**:393-395 (June) 1928.

79. Kopp, M. M., and Levy, H.: Foreign Body in the Nose (Report of a Case), Laryngoscope **48**:819-821 (Nov.) 1938.

80. Gutherie, D.: Foreign Bodies in the Nose, J. Laryng. & Otol. **41**:454-457 (July) 1926.

81. Clerf, L. H.: Historical Notes on Foreign Bodies in the Air Passages, Ann. Med. Hist. **8**:547-552 (Nov.) 1936.

82. Killian, G.: The Treatment of Foreign Bodies in the Respiratory Tract and Esophagus, Ann. Otol. Rhin. & Laryng. **16**:251-287 (June) 1907.

83. Goldman, J. L.: Fish Bones in the Esophagus, Ann. Otol. Rhin. & Laryng. **60**:957-973 (Dec.) 1951.

84. Boyd, G.: Esophageal Foreign Bodies, Canad. M. A. J. **64**:102-107 (Feb.) 1951.

85. Jackson, C., and Jackson, C. L.: Bronchoesophagology, Philadelphia, W. B. Saunders Company, 1950, pp. 13, 15, 25, 261, 259, and 260.

86. Matheson, I.: Foreign Bodies in the Esophagus: A Review of 602 Cases, J. Laryng. & Otol. **63**:435-445 (Aug.) 1949.

87. Norris, C. M.: Foreign Bodies in the Air and Food Passages: A Series of 250 Cases, Ann. Otol. Rhin. & Laryng. **57**:1049-1071 (Dec.) 1948.

88. Holinger, P. H.; Andrews, A. H., Jr., and Rigby, R. G.: Foreign Bodies in the Air and Food Passages: Report of a Series of 525 Cases, Eye, Ear, Nose & Throat Month. **22**:415-424 (Nov.) 1943.

89. Clerf, L. H.: Foreign Bodies in the Air and Food Passages: Observations on End-Results in a Series of 950 Cases, Surg., Gynec. & Obst. **70**:328-339 (Feb.) 1940.

90. Wrigley, F. G.: Series of 200 Cases of Esophagoscopy, Brit. M. J. **2**:334-335 (Aug 12) 1939.

91. McCannel, A. D.: Peroral Endoscopy (Observations in 200 Cases of Foreign Bodies), Canad. M. A. J. **37**:42-47 (July) 1937.

92. Cummings, G. O.: One Hundred Seventy-Nine Foreign Bodies in the Food Passages, Laryngoscope **46**:227-236 (March) 1936.

93. Clerf, L. H.: Safety Pins in the Esophagus, Arch. Otolaryng. **24**:282-288 (Sept.) 1936.

94. Jackson, C., and Jackson, C. L.: Diseases of the Air and Food Passages of Foreign Body Origin, Philadelphia, W. B. Saunders Company, 1936, pp. 1-636; 36-38; 26-28; 39.

95. Manges, W. F.: The Roentgenology of Foreign Bodies in the Esophagus and Gastro-Intestinal Tract, S. Clin. North America **14**:89-95 (Feb.) 1934.

96. Vinson, P. P.: Foreign Bodies in the Air and Food Passages: Report Based on 334 Cases, M. Clin. North America **16**:1471-1478 (May) 1933.

97. Lerche, W.: The Esophagoscope in Removing Sharp Foreign Bodies from the Esophagus, J. A. M. A. **56**:634-637 (March 4) 1911.

98. Marmor, W. A.: The Missouri Sales Tax Token as a New and Relatively Common Foreign Body Requiring Removal from the Esophagus and Pharynx (31 Cases), J. Missouri M. A. **37**:293-294 (July) 1940.

99. Liu, J. H.: Foreign Bodies in the Esophagus, Chinese M. J. **43**:556-563 (June) 1929.

100. Reading, P.: Some Observations on the Etiology of Foreign Bodies in the Esophagus, J. Laryng. & Otol. **60**:246-249 (June) 1945.

101. Lees, K. A.: Coins in the Esophagus, Brit. M. J. **1**:761 (April 23) 1927.

102. Holinger, P. H.; Johnston, K. C., and Greengard, J.: Congenital Anomalies of the Esophagus Related to Esophageal Foreign Bodies, Am. J. Dis. Child. **78**:467-476 (Oct.) 1949.

103. Landers, J. J.: Observations on Foreign Body Swallowing in Prisons, J. Ment. Sc. **95**:897-900 (Oct.) 1949.

104. Schwartz, V. J.: Thermometer as a Foreign Body in the Esophagus, J. A. M. A. **110**:953 (March 26) 1938.

105. Arbuckle, M. F., and Stutsman, A. C.: Safety Pin in the Alimentary Tract: Diagnosis, Treatment, Dangers, Complications, South. Surgeon **7**:131-135 (April) 1938.

106. Stewart, N. E.: Method to Promote Passage of Foreign Bodies Swallowed in Attempted Suicide (Case), M. Bull. Vet. Admin. **7**:1082 (Nov.) 1931.

107. Imperatori, C. J.: An Unusual Foreign Body, Arch. Otolaryng. **11**:213-214 (Feb.) 1930.

108. Yankauer, S.: Thirteen Foreign Bodies Removed from the Esophagus at One Sitting, Arch. Surg. **12**:447-448 (Jan.) 1926.

109. Randall, H.: Foreign Body in the Esophagus of an Infant, Laryngoscope **38**:545 (Aug.) 1928.

110. Chamberlin, W. B.: Dysphagia of Septic Origin Resembling a Foreign Body in the Esophagus, Laryngoscope **41**:36-38 (Jan.) 1931.

111. Clerf, L. H.: Foreign Bodies in the Esophagus, Ann. Otol. Rhin. & Laryng. **35**:1000-1020 (Dec.) 1926.

112. Kreutz, G. C., and Dill, J. L.: Nonopaque Foreign Bodies in the Esophagus, with Report of 4 Cases, Am. J. Surg. **46**:532-534 (Dec.) 1939.

113. Rashid, A.: Atypical Case of Foreign Body in the Esophagus, Laryngoscope **60**:945-946 (Sept.) 1950.

114. Fabritius, H. G.: Foreign Bodies in the Esophagus with Symptoms Exclusively from the Air Passages, Acta Pædiat. **37**:335-340, 1949.

115. Stevenson, R. M.: X-Ray Investigation of Stridor (Due to Foreign Body in the Esophagus) in a Child, Brit. M. J. **1**:221 (Feb. 20) 1943.

116. Friedberg, S. A.: Esophageal Foreign Body with Symptoms Suggesting Other Thoracic Disease, Arch. Otolaryng. **34**:591-592 (Sept.) 1941.

117. Tucker, G.: Jackstones in the Upper Food Passages: Reports with Esophagoscopic Observations on 11 Cases, Ann. Otol. Rhin. & Laryng. **46**:237-247 (March) 1937.

118. Jackson, C., and Jackson, C. L.: Pulmonary Symptoms Due to Esophageal Disease, Arch Otolaryng. **18**:731-745 (Dec.) 1933.

119. Thacker-Neville, W. S.: Foreign Body in the Esophagus Causing Dyspnea, J. Laryng. & Otol. **43**:105-106 (Feb.) 1928.

120. Naftzger, J. B., and Gittins, T. R.: Foreign Bodies in the Esophagus with Respiratory Symptoms Complicating Diagnosis: Report of 4 Cases, Laryngoscope **36**:370-376 (May) 1926.

121. Friedberg, S. A.: Foreign Bodies in the Esophagus, Ann. Otol. Rhin. & Laryng. **26**:768-781 (Sept.) 1917.

122. Weaver, D. F., and Moersch, H. J.: Esophageal Obstruction Due to Persimmons: Report of a Case, Proct. Staff Meet., Mayo Clin. **13**:129-131 (March 2) 1938.

123. Iglauer, S.: Nonopaque Foreign Bodies in the Esophagus and Esophagotracheal Fistula: Their Demonstration by Ingested Oil, Arch. Otolaryng. **7**:229-233 (March) 1927.

124. Iglauer, S.: Ingested Lipiodol to Demonstrate Nonopaque Foreign Bodies in the Esophagus, Ann. Otol. Rhin. & Laryng. **36**:1157-1161 (Dec.) 1927.

125. Skarby, H. G.: Clinical and Roentgenologic Diagnosis of Foreign Bodies, Especially Fish Bones, in the Hypopharynx and Esophagus: Technique of Roentgen Examination, Acta radiol. **25**:796-824, 1944.

126. Head, J. R.: Surgical Indications in Perforation of the Esophagus by Foreign Bodies, Am. J. Surg. **42**:266-274 (Oct.) 1938.

127. Latimer, M. L.: Impacted Foreign Bodies in the Esophagus, Arch. Otolaryng. **29**:550-552 (March) 1939.

128. Laff, H. I.: Fishbone in the Mediastinum Removed by Esophagoscopy, Ann. Otol. Rhin. & Laryng. **59**:690-693 (Sept.) 1950.

129. Strieder, J. W.: Surgery of the Esophagus, New England J. Med. **243**:445-454 (Sept. 21) 1950.

130. Jemerin, E. E., and Aronoff, J. S.: Foreign Body in Thyroid Following Perforation of Esophagus, Surgery **25**:52-59 (Jan.) 1949.

131. Barrie, H. J., and Townrow, V.: Perforation of the Aorta by a Foreign Body in the Esophagus: Case Report and Review of the Literature, J. Laryng. & Otol. **61**:38-42 (Jan.) 1946.

132. Kearney, H. L.: Fatal Hemorrhage from Penetration of the Right Renal Artery by a Fish Bone, Ann. Otol. Rhin. & Laryng. **53**:797-799 (Dec.) 1944.

133. Simons, J.: Traumatic Ulceration of Aorta, Brit. M. J. **1**:233 (Feb. 5) 1927.

134. Moore, P. M., Jr.: Esophagoscopy: Treatment of Lesions of the Esophagus, Cleveland Clin. Quart. **7**:52-57, 1940.

135. Saltzstein, H. C.: Duck Bone Impacted in Lower End of Esophagus: Erosion of Esophageal Wall; Fatal Hemorrhage, Ann. Surg. **95**:794-797 (May) 1932.

136. Harvey, G., and Davy, A. O.: Foreign Body in Esophagus Associated with Massive Collapse of Lung, M. J. Australia **1**:148-149 (Feb. 2) 1935.

137. Miller, J. W.: Esophageal Perforation: Two Fatal Cases, New York J. Med. **37**:2093-2095 (Dec. 15) 1937.

138. Arens, R. A., and Stewart, E.: Pneumopericardium Following Foreign Body in the Esophagus, Radiology **22**:334-338 (March) 1934.

139. Imperatori, C. J.: Pericarditis as a Terminal Lesion in Mediastinitis: Report of a Case, Following the Inhalation of a Foreign Body in the Bronchus and Accompanied by Spontaneous Emphysema, Ann. Otol. Rhin. & Laryng. **42**:923-926 (Sept.) 1933.

140. Watson-Williams, E.: Overlooked Foreign Body in Esophagus: Retro-Esophageal Abscess, Laryngeal Edema; Tracheotomy; Septicemia, Death, J. Laryng. & Otol. **52**:179 (March) 1937.

141. Thacker-Neville, W. S.: Foreign Body in Esophagus with Streptococcal Septicemia and Mediastinitis, Brit. M. J. **1**:978-979 (May 13) 1939.

142. Wrigley, F. G.: Case of ? Swallowed Foreign Body Resulting in Meningitis, J. Laryng. & Otol. **47**:249-251 (April) 1932.

143. Meulen, P. V. D.: Besondere Komplikationen durch einen Fremdkörper in der Speiseröhre, Acta oto-laryng. **30**:481-486, 1942.

144. Jackson, C. L.: Foreign Bodies in the Air and Food Passages, Post-Grad. M. J. **4**:281-290 (Oct.) 1948.

145. Palmer, E. D.: The Esophagus and Its Diseases, New York, Paul B. Hoeber, Inc., 1952, p. 192.

146. Clerf, L. H.: Historical Aspects of Foreign Bodies in the Air and Food Passages, Ann. Otol. Rhin. & Laryng. **61**:1-17 (March) 1952.

147. Gudger, E. W.: Live Fishes Impacted in the Pharynx of Man, Arch. Path. & Lab. Med. **4**:346-355 (Sept.) 1927.

148. Fishman, L. Z.: Mechanism Involving Foreign Bodies in the Postcricoid Narrowing, with Special Reference to Sharp Foreign Bodies Such as Open Safety-Pins, Arch. Otolaryng. **17**:747-754 (June) 1933.

149. Latella, P. D., and Jones, D. H.: An Unusual Course of an Open Safety Pin in a Baby 8 Months Old, Ann. Otol. Rhin. & Laryng. **59**:254-257 (March) 1950.

150. Myerson, M. C.: Foreign Bodies in the Stomach and Intestines, Arch. Otolaryng. **8**:675-685 (Dec.) 1928.

151. Schwartz, V. J.: Unique Case of Foreign Body in the Esophagus, Arch. Otolaryng. **45**:562-567 (May) 1947.

152. Jesberg, S.: Disks in the Esophagus, Arch. Otolaryng. **11**:210-213 (Feb.) 1930.

153. Withers, B. T.: Foreign Bodies of the Larynx with Report of an Unusual Case, Ann. Otol. Rhin. & Laryng. **58**:1085-1093 (Dec.) 1949.

MODERN ASPECTS OF THE RECRUITMENT PHENOMENON

MARTIN P. LANSBERG, M.D.
SOESTERBERG, NETHERLANDS

TABLE OF CONTENTS

I. Introduction
II. Recruitment and hearing aids
III. Recruitment and neural lesions
IV. Partial Recruitment
A. In severe lesions
B. In conductive lesions
1. The normal impedance function
2. The pathological impedance function
3. The dynamogram
4. Loudness in bone conduction
5. The 2,000 bone conduction dip in otosclerosis
C. In mixed types of deafness
V. Recruitment in cases of herpes oticus
VI. Recruitment in multiple sclerosis
VII. Testing technique
A. Older techniques
B. Hood's perstimulatory test
C. Lüscher's test
VIII. Comment
A. General considerations
B. Acoustic trauma
C. Ménière's disease
D. Fatigue
E. Eighth-nerve lesion
IX. Conclusions
X. Summary

I. INTRODUCTION

FEW ITEMS in modern audiological research have attracted so much interest as the recruitment phenomenon. The story of its still young history reveals many remarkable features. It will be the purport of this paper to discuss the fallacies of recruitment testing, a routine which has of late become an indispensable aid in clinching many of our diagnoses. A consideration of these fallacies will at the same time bear in a broader sense on our concepts of hearing mechanisms.

When Fowler Sr.[3] became interested in the above-threshold testing, his idea was that early diagnosis of otosclerosis might be achieved by this method. The argument was that slight interference with stapes mobility would be demonstrated at low sound pressures and be overcome by increasing the sound pressure; a threshold loss was expected, with normal loudness sensation at higher levels. The swifter than normal rise in loudness became designated as loudness recruitment. As so often has happened with brilliant ideas in medical research, the outcome proved different from what was anticipated. The result of inspiration and perspiration nevertheless was very satisfying, since out of these efforts came a new and better understanding of auditory function. It was found that no impedance lesion could be detected by this test; there was no such thing as a mechanical barrier for

From the Ear, Nose, and Throat Department, National Aero-Medical Center.

Statements and conclusions expressed in this paper do not necessarily reflect the opinion or policy of the National Aero-Medical Center.

tones of low sound pressure giving way at higher outputs. Contrary to expectations, it was perception deafness that revealed the recruitment factor frequently and in an unmistakable fashion. The idea had proved to be a fruitful one. Yes, an even more intriguing quality of this recruitment—the so-called overrecruitment —was added to the mystery of the newly found phenomenon.

Both in the old and in the new world many investigations followed the appearance of the original report. Of all the literature on the subject which I have perused, the thesis by de Bruïne-Altes [2] should be mentioned for its completeness in dealing with the subject clinically and theoretically and for its introduction of new methods of demonstrating the effect (monaural balance technique).

II. RECRUITMENT AND HEARING AIDS

A second unexpected clinical aspect of the recruitment factor arose when speech intelligibility was investigated. Speech being "suprathreshold" in character was at "prima vista" expected to be in a relatively favorable position in cases of recruitment deafness. Once the threshold loss was compensated for, hearing would be as good as in normal ears. It soon became clear, however, that recruitment was the most troublesome burden in achieving proper speech intelligibility. As is now known, this must be ascribed to the sound distortion accompanying recruitment deafness. This distortion results from the following factors:

A. Dynamic dysacousis is due to the speech pattern being torn apart in a vertical way. Dynamic dysacousis is the recruitment phenomenon *sensustrictioi*: "An accelerated loudness sensation versus sound pressure function."

B. Diplacusis gives discrepancy in right-left pitch impression.

C. Disharmonic paracusis gives a nebulous and unpleasantly foggy twang to the sounds perceived.

D. Self-masking also makes speech discrimination very vulnerable.[37] Self-masking is auditory fatigue; the term is confusing because it has nothing to do with the ordinary masking of one tone by an other; self-masking is an after-effect designating the abnormally large amount of fatigue in cases of recruitment.

Thus it became clear that recruitment was a great handicap in restoring good hearing. Mention must be made here of the investigations by Harris and his collaborators.[1] They demonstrated that loudness of speech runs parallel with loudness of pure tones; this is exactly what should be expected, since the intelligibility of speech, as pointed out above, is only to a certain degree determined by the loudness and is largely dependent on other factors. This was beautifully shown by Huizing and Reyntjes.[42]

III. RECRUITMENT AND NEURAL LESIONS

A third revelation in this enchanted labyrinth came from Hallpike and his collaborators.[4] In a series of cases of Ménière's disease and eight-nerve tumors, the former showed a positive recruitment, whereas the latter did not. The conclusion to be deduced from these findings was that a differential diagnosis between end-organ lesions and neural lesions could be based on a functional test. The ingenuity of Hallpike, had quite unexpectedly, added new glamour to above-threshold phenomena; new interest was evoked in investigating recruitment symptomatology.

The clinical importance was evident. It is fair to remind the reader of the absence of recruitment found several years before by de Bruïne-Altes in cases of presbycusis which implied that fundamental differences existed in cases of perceptive deafness of different origin. Hallpike's findings were moreover substantiated by later investigators (Eby and Williams [83]). These authors also report absence of diplacusis in eighth-nerve tumors and a speech intelligibility curve shifted upwards but otherwise normal in its course, whereas end-organ lesions show a distorted intelligibility curve with a maximum instead of the normal asymptotic "S" shape.

IV. PARTIAL RECRUITMENT

Little mention has been made in the foregoing account of the amount of recruitment. Yet in clinical testing this is an important factor, since full recruitment or even overrecruitment is not necessarily present. What is one to conclude in these cases in which only partial recruitment is present? Diagnosis can be burdensome and fraught with technical problems taxing one's audiometric ability.

A. Partial Recruitment in Severe Lesions.—For instance, in cases of great monaural loss a balance test can simulate the absence of recruitment, whereas overhearing is the cause of this effect. Especially in cases of some 90 db. loss the diagnosis between inner-ear and neural deafness becomes very important. When, without masking, in such circumstances I find a loss of 60 db. and start my binaural balance procedure, it is evident that no recruitment will be found, even if in reality the lesion is a cochlear one. With provision on the audiometer for outputs above 100 db. one might then, however, expect to find the phenomenon, but testing once again becomes difficult at these levels due to its unpleasantness and even painfulness. When masking procedures are introduced at these high levels, they should be handled and interpreted with great carefulness.

B. Partial Recruitment in Conductive Lesions.—Another possibility of partial recruitment—which I especially intend to present here—is frequently found in cases of middle-ear impairment, i. e., those caused by serous otitis media or adhesive processes in the cavum tympani. According to the theory outlined above, I was reluctant at first to consider my findings correct. However, the phenomenon could be so obviously demonstrated in many cases that an explanation had to be given. This explanation, it seems to me, is readily at hand and has, till the present, only been veiled by a misunderstanding of the normal loudness curve.

1. The Normal Impedance Function: I will attempt to retrace what happens in a normally functioning ear with loudness perception: No fixed relation exists between sound pressure and subjective loudness; the Weber-Fechner law is not in accord with the Fletcher-Munson loudness contours,[5] neither for the various frequencies nor for the various outputs. Indeed at higher outputs the loudness increase is accelerated. Can this be understood in a mechanical way? To explain this with the impedance formula, as was done by Mygind [6] and Veckmans,[7] cannot satisfy our physical reasoning; that tones should have great reduction of this impedance at higher sound levels can never be reasonably argued on theoretical grounds for the very complicated middle-ear and inner-ear mechanisms. On the contrary, the microphonic cochlea effect has proved that a purely linear function exists between

input and output up to the locus of distortion. Any diversion from this linear function cannot take place beyond the locus of distortion. Faster than linear increase of loudness impression therefore starts in or proximal to the cochlea.

Loudness perception is an over-all function and finally a psychological one; sensorial and neurophysiological factors play their part in the loudness intensity function, and certainly density of hair cells and availability of nerve fibers (for different frequencies increasing in different and unproportional degrees) can partly contribute to an understanding of loudness function, but this is central to the region in which impedance relations prevail (Wever [8]).

Mechanical impedance does not reach further than the inner-ear fluids. Mechanical impedance, also, is for all practical purposes minimal at low sound pressures.* Any extra increase in loudness cannot be explained by impedance changes. On the contrary, I know that the middle-ear muscles can only add to the impedance and that the result is a loudness reduction at higher levels. When, secondly, I look for experimental evidence of what impedance fluctuations at different sound frequencies can do, I recall the experiments performed with the acoustic bridge by Metz [11] and Kristensen.[12] As Bornschein and Krejci [13] aptly point out, these experiments can only answer our curiosity as to sound absorption in the peripheral part of the middle ear, but no conclusions can be derived with this technique for the sound transmission to the ear. Sound absorption and sound transmission do not run parallel courses under changing conditions. Indeed the relations are entirely different, as is shown by the electrocochleographic registrations of Bornschein and Krejci.[13] True, they demonstrate the considerable influence that muscular contractions in the middle ear have in increasing the impedance, but certainly this increase is no less for the lower frequencies than for the higher ones, and, consequently, no relative enhancement of the lower frequencies at higher sound levels can be explained on this basis. In earlier experiments by Wever and Bray [14] the same relation was found: a greater influence on lower frequencies, consequently, a greater rise in impedance and more reduction of the sound transmission for the lower frequencies.

These findings show that a fixed selective rise in impedance does not exist and that higher frequencies are damped to a nearly similar extent at greater outputs as are the lower frequencies. That higher frequencies should, however, be more influenced by muscular spasm than the lower ones can certainly never be inferred from any of the experiments. Just as the disproportionate rise in loudness at high outputs, the disproportionate rise in loudness for the lower frequencies over the higher ones must find its explanation in central mechanisms: sensorial, neural, and psychological factors—all proximal to the entrance of sound in the cochlea.

2. The Pathological Impedance Function: After this elaboration on normal loudness relations, my object, explaining the recruitment in cases of middle-ear impairment, will be continued. The explanation now seems to be at hand and can be formulated in the following question: Will contraction of the middle-ear muscles effect the same impedance increase in the transmission-defective ear as in the normal ear? Or, to formulate the explanation in a more general way: Are impedance relations in the sound transmission apparatus equally influenced by changes in both normal and transmission-defective ears? The answer, obviously, is no. Then

* References 9 and 10.

why is this difference not more often found? Again the answer is evident: 1. The influence of muscular contraction in the middle ear only appears at higher sound levels and, consequently, will in loudness matching only be demonstrable at 60 db. or more above the threshold; one usually does not investigate at outputs so much above threshold, because it means 60 db. or more above the defective threshold. 2. The effect in terms of decibels is relatively small.

The largest difference between normal and transmission-defective ears that one can expect to find reflects a maximal rise in impedance for the normal ear and no rise at all for the defective one. Many are the possibilities of transmission disturbances, and it certainly is only a minority of cases that both the stapedial and the

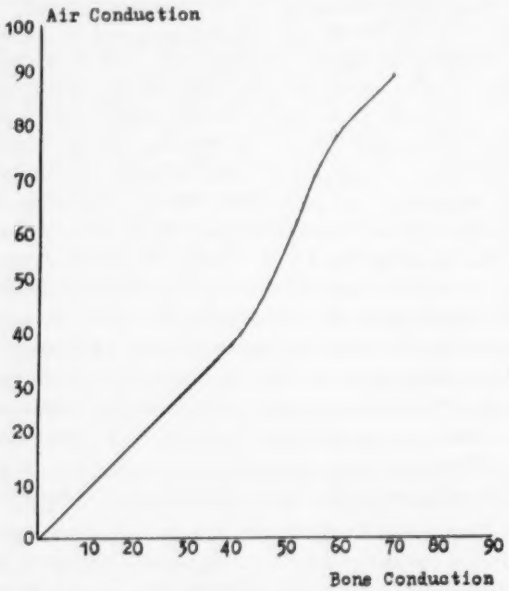


Fig. 1.—The graph represents the loudness relation between air conduction and bone conduction for a 500-cycle tone. A departure from linearity starting at 45 db. can be clearly seen.

tensor muscles are out of action. Nevertheless, one should not expect normal impedance-changing behaviors in all cases in which these muscles are intact. On the contrary, the effect of these muscular contractions will in all probability only appear when they work under normal conditions.

Clinical evidence on this point comes from van Dishoeck's [88] investigations with the pneumophone. With this instrument changes in sound transmission are brought about by applying positive or negative pressure in the outer-ear canal, thus changing the stiffness of the conduction apparatus. A reduction in sound transmission, as judged by a reduction in subjective loudness, originates under these circumstances. No such reduction from increased stiffness will, however, be found when the cavum tympani is filled with fluid (serous otitis). Although stiffness alterations due to muscular contractions will not be exactly the same as stiffness

alterations due to positive or negative pressure, the general principle that an increase in impedance, resulting from increased stiffness, only appears when the cavum tympani is normal and filled with air remains valid.

Thus I feel completely confident in finding recruitment in cases of serous otitis or otosclerosis in which the middle-ear muscles are not affected. It is my aim to investigate these relations experimentally in electrocochleographic recordings. An important aspect of the given explanations is the type of loudness curve that one may expect in transmission-defective ears; there is no recruitment at intensities little above threshold, the phenomenon appearing at some 60 db. above this level (Fig. 2).

One more question: Is one justified in using the word recruitment in these cases? What actually happens is the matching of an ear lacking the capacity to change its

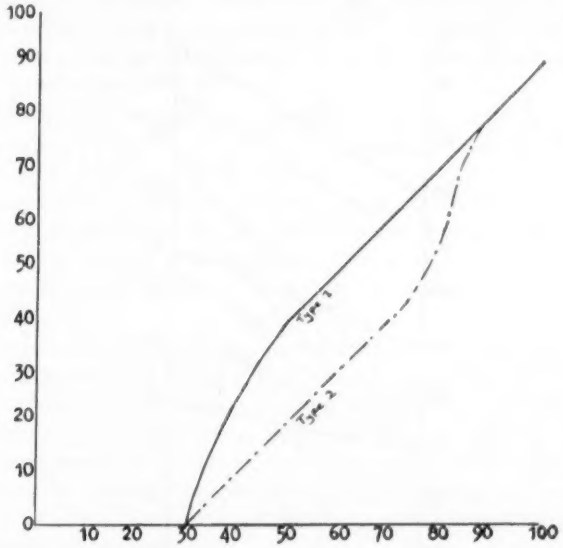


Fig. 2.—Type 1 is a loudness curve for a case of mixed deafness with a 30-db. threshold loss, of which 20 db. originates from a cochlear lesion. Type 2 symbolizes a purely conductive lesion, as, e. g., in a case of serous otitis.

mechanical impedance with a normal one that possesses this function. Thus it is an extra potency of the normal ear, comparable to the well-known automatic volume compression in hearing aids, that is described in terms of a special quality or rather defect of the diseased ear. I do not object, however, to the application of the word recruitment in these cases also, because, as I have stated before, loudness is a complex function which finally is of psychological character. Are there more indications and practical implications of the recruitment playing a part in transmission types of deafness? I think so.

3. The Dynamogram: Mol † described in cases of otosclerosis an accelerated loudness function between the well-known levels of comfort and discomfort named by him a dynamogram. This easily fits in with my theories given above; the absence of volume compression is an indication of recruitment in otosclerotic ears.

† References 15 and 16.

4. Loudness in Bone Conduction: A second clinical item to be discussed here is the well-known loudness matching between air conduction and bone conduction. A recruitment was found for bone conduction which was not explained for by the theory and therefore not tolerated; as it happens in those instances, a most unsatisfactory semiexplanation was given by stating that this effect was due to the testing technique in improperly sound-proofed rooms. That this negation of the real problem is unsatisfactory will be clear to anyone who is working in properly sound-isolated environments.

The real reason lies once again in the different influence that impedance-changing factors have on air-conducted and bone-conducted sounds. From a series of experiments performed in my acoustical laboratory I found relations, as graphically

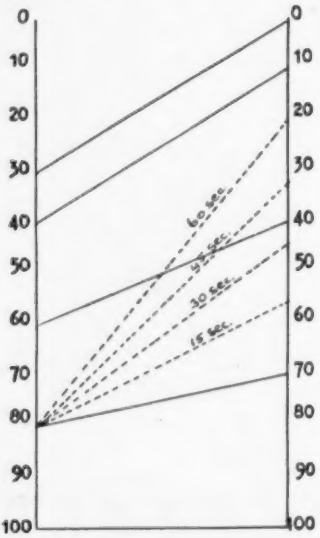


Fig. 3.—A diagrammatic representation of Hood's perstimulatory fatigue in a case of Ménière's disease. Further explanation is found in the text.

given in Figure 1, for a tone at 500 cps (Hz). It is shown in this graph that the air conduction-bone conduction balance creates a 45-degree slope—thus equal loudness increment—up to 45 db. Then with a sharp rise the bone conduction exceeds the air conduction by 20 db. At this point the activity of the middle-ear muscles comes into play to an extent which conforms well with my other findings.

If the same test is performed for a 2,000-cycle tone, one does not find this departure from linearity. Does this mean that no influence of muscular activity exists for this frequency, as has been thought (this theory was refuted by Wever's as well as by Bornschein's experiments)? As mentioned above, I do not believe so. I feel that the other possibility, namely, a mainly identical influence on both air conduction and bone conduction, exists as a result of muscular contractions for this tone, whereas for the 500-cycle tone hardly any effect on bone conduction due to muscle spasm is to be expected. Harris [1] states that he could not find the effect; however,

in his graph a rise at 45 db. for the 500-cycle tone can clearly be seen; it is exactly at this point that I found it. Harris stopped at this level and thereby obviously missed the crux of the phenomenon.

I shall elaborate on this point a little further, since the evidence that will be brought forth has become of great practical importance. Experiments on the cat by Kendon Smith [19] have shown an unmistakable influence of stapedial fixation on bone conduction. Alas, his results show that the influence is preponderant in the middle frequencies (300 to 1,800) and negligible at 2,000 d.v.

It must be remembered that what I want to investigate is the difference in influence that stapedial fixation has on air conduction and on bone conduction, anticipating a great difference at low but hardly any difference at high frequencies. From Smith's tracings I counted at 300 d.v. an air-conduction reduction of 30 db. and a bone-conduction reduction of 5 db., a difference of 25 db. I counted at 2,000 d.v. an air-conduction reduction of 21 db. and a bone-conduction reduction of 1 db., a difference of 20 db.

Thus, from Smith's experiments one might compute a 25-db. recruitment for bone conduction at 300 d.v. and a 20 db. recruitment for bone conduction at 2,000 d.v. The 25 db. at 300 cycles corresponds rather well with my findings; the 20 db. at 2,000 cycles, however, does not. This may, to my mind, be reasonably attributed to the abnormal experimental circumstances (opened bulla and thread tied around the stapes) and to the fact that anatomical differences between the cat's ear and the human ear make it inadmissible to transfer animal findings in absolute terms to human relations.

Are there grounds to substantiate this hypothesis? In other words, can clinical or experimental evidence be found that will support my theory of selective impedance relations in bone conduction as a function of frequency? The angle from which I shall approach this problem seems different from my present meditations on recruitment phenomena, which are all based on suprathreshold measurements. As I now introduce the threshold reading of otosclerotic deafness, it might seem that I am journeying a long way from my starting point. I hope that the reader will join me in my excursion.

5. The 2,000 Bone Conduction Dip in Otosclerosis: On closer investigation I realize that what I want to prove is that sound transmission in bone conduction is influenced for certain frequencies by the prevailing properties of the middle-ear mechanism. I want to give evidence that changing or changed mechanical relations in the middle ear have their repercussion for certain frequencies also on bone conduction. Whether I choose to substantiate this hypothesis by the physiologically occurring stiffening of the muscles at above threshold level or the pathologically occurring stiffening or even ankylosis of the stapedial joint in cases of otosclerosis, remaining equal at all sound levels, is immaterial. In the preceding paragraphs I concluded from my above threshold balancing tests that the 2,000 bone conducted frequency shows an impedance increase with stiffening of the middle-ear mechanism. In the bone-conduction audiogram of otosclerosis I found a similar effect in the form of a 2,000 dip. Can this finding bear out my theory or is this 2,000 dip due to an inner-ear lesion? The practical importance of this last question occurred to me in 1947, when I became interested in the selection of patients for fenestration operation. If this dip would betray and parallel that of an inner-ear

lesion to the same extent, many otosclerosis cases would have been rejected and the operation withheld because reaching the sociable level of hearing would *a priori* be impossible.

In a paper read before the Dutch Ear, Nose, and Throat Society in 1947,[43] I described the dip and discussed its origin. Postoperative audiograms showed that this dip could not represent in full an inner-ear lesion, and in preoperative testing I could indeed demonstrate with a special technique (intermittent binaural balance for different frequencies) that the dip was largely due to an impedance lesion. In predicting results to be anticipated from fenestration, I therefore set great store by the bone-conduction reading at higher frequencies—4,000 and even 8,000 cycles—since if these were normal, no trouble was to be feared. To prove my point at that time I imitated a stapedial fixation by experimenting on patients without a drum membrane and two outer ossicles. A bone-conduction telephone was placed on the mastoid, and with a tiny little hook pressure or tension was exerted on the stapes. The result was in accordance with my expectation, since a depression in sensitivity could clearly be demonstrated for 2,000 cycles. The experiment is rather difficult, and no exact quantitative data were obtained, but the effect was undeniable. The same effect on bone-conduction sensitivity for the region of 2,000 d.v. was described by Groen [20] in the following words (a verbatim translation): There is a clear, albeit often small (10 to 20 db.) bone-conduction loss around 1,000 to 3,000 db. For the proper appreciation of this statement of Groen's, it is pertinent to remember that he was discussing impedance changes. A thorough and extensive clinical investigation was made by Carhart.‡ Thus the 2,000 bone-conduction notch described by Lansberg, Groen, and Carhart became firmly established as an impedance lesion in bone conduction. Whether the inertia or the compression factor of bone conduction or both these factors cause the dip will not be discussed here.

After this short exposé on bone-conduction readings in otosclerosis, I can return to my starting point, which was the finding of recruitment in bone conduction-air conduction matching. It has by now become evident that the explanation should be interpreted in the following terms: The automatic volume compression normally occurring in air conduction at levels above 50 db. finds no corresponding effect in bone conduction for most frequencies; at 2,000 cycles, however, the influence of stiffening of the middle-ear joints results in an impedance increase which, to a major extent, equals the automatic volume compression in air conduction. In the latter case air conduction-bone conduction balancing will show no disproportionate loudness increase in favor of bone conduction, whereas at 500 d.v. it does and consequently may be interpreted as loudness recruitment for bone conduction. Absence of automatic volume compression means presence of recruitment.

C. Partial Recruitment in Mixed Types of Deafness.—Partial recruitment will also be found in combined lesions of neural and cochlear origin or of cochlear and peripheral origin. As opposed to the recruitment described above—as a consequence of wanting the natural automatic volume compression—the recruitment in the mixed lesions will show another course. In Figure 2 a diagrammatic representation of these two types is given, and it can be seen that the course taken in mixed deafness is just the opposite of Curve 2. In Curve 1 a 30 db. loss is represented as being due to 10 db. neural and 20 db. cochlear loss; the 20 db. loss due to this cochlear lesion

‡ References 21 and 22.

is quickly balanced out a little above threshold values. Conversely, the 30 db. loss in a case of serous otitis will show its lack of automatic volume compression only at those intensities where this impedance-increasing factor shows itself, which is at some 50 db. or more above threshold. This might seem to be an easy way of differentiating on functional grounds two types of deafness; indeed it is possible, when carefully searched for, to support an etiological diagnosis on these grounds. Every otologist who is familiar with balance testing for recruitment knows, however, of the difficulties in exact quantitative matching—difficulties arising from duration of tone exposure, dysacousis, diplacusis, fatigability, the sequence of offering the tone first to the diseased and then to the normal ear or in the reversed way, and several more. These factors will be dealt with more extensively in the section on Testing Technique. All these factors make it rather hazardous to build a diagnosis on these small differences.

V. RECRUITMENT IN CASES OF HERPES OTICUS

The syndrome described by Ramsay Hunt falls, much to my regret, more often in the able hands of neurologists than in those of otologists; with due deference to my highly esteemed colleagues and friends who investigate the most delicate system of the human body with their reflex hammer, I feel that cochlear and labyrinthine testing techniques can, as a rule, be better dealt with and more extensively explored by otologists. Close cooperation, however, between neurological and otological departments can often elucidate diagnostic and theoretical problems which otherwise would remain in the twilight between knowledge and ignorance. This statement of more general validity applies, it seems to me, also to the Ramsay Hunt syndrome. Neurological research has given the answer to the central nervous lesion being a ganglionitis or even a medullitis.[28] In a case that I had occasion to investigate I found nearly total recruitment. Does this agree with Hallpike's theory of recruitment being present in cochlear and absent in neural lesions? At first glance I should say no, because the lesion in herpes oticus is central to the cochlea. However, is it extravagant to reverse the *onus probandi* and tentatively propose the theory that in herpes oticus the ectodermal structures of the scala media undergo similar changes as occur in the skin in cases of herpes zoster, changes from which this disease derives its name? Let me indulge in my premises and fantasies a little further: a hydrops of the scala media might originate, and a situation might result largely comparable to Ménière's disease.

Lempert,[84] in a recent study, has even gone so far as to contend that Ménière's disease is a chronic progressive herpetic neuritis. In the scope of this article no further elaboration can be relevant to my problem. I am awaiting further evidence from a number of cases, but for the critical reader I can quite definitely state that the recruitment in my case could in no way be explained by loss of stapedial function due to facial paralysis. There was complete recruitment of some 60 db. In the meantime I wish to thank Dr. Stenvers, who investigated the patient neurologically. His diagnosis of the case wavers between herpes oticus and arachnoiditis; Stenvers suggests this latter possibility because he has not seen the skin eruption and the rapidly vanishing inflammatory lesion on the tonsillar mucous membrane.

Arachnoiditis would, it may be mentioned here, be still more inconsistent and unexplainable with the complete recruitment, since the acoustic channels may be damaged from arachnoiditis only in the neural and certainly not in the cochlear part.

Hallpike § finds, as I, a large recruitment in herpes oticus; he ascribes this to a serous meningitis. I wonder whether this could explain the complete recruitment, even if it is supposed that the serous meningitis would have a corollary in a serous labyrinthitis of the perilymphatic fluids. Then why should not this happen in other cases of serous meningitis? An eruption in the epidermal structures of the scala media still appeals more to me as the probable cause. Parenthetically it may be mentioned here that in my opinion many cases of otitic herpes remain undiagnosed, especially in cases in which there is no facial paralysis; the sudden deafness often combined with pain and eruption in the outer-ear canal is, as I see it, due to herpes infection, just the same as in Hunt's syndrome. I have seen several cases of this type.

VI. RECRUITMENT IN MULTIPLE SCLEROSIS

Some four years ago I tried to find out whether recruitment existed in cases of multiple sclerosis that showed a hearing loss. Much to my regret I could not prove to my full satisfaction that recruitment was absent, which was largely due to the fact that hearing losses were small and testing was difficult. Unfortunately, no support could therefore be found from this angle for my present conviction that neural lesions don't show recruitment. Von Leden and Horton,[24] in a study on deafness in multiple sclerosis, alas, did not mention recruitment testing; they state, however, that they found absence of diplacusis, which, although not obligatory, is a faithful companion of recruitment. Absence of diplacusis forms strong presumptive evidence of absence of recruitment.

VII. TESTING TECHNIQUE

A. Older Techniques.—It is not my aim to dwell on the well-known older techniques, of which I mention the binaural balance (two ears and one frequency), the monaural balance (one ear and two frequencies), and monaural masking. In speech range determinations the range between the speech reception threshold, the most comfortable level, and the level of discomfort, which constitutes the span of speech intelligibility, is narrowed in cases of recruitment.‖ It will be remembered that the term dynamogram was coined by Mol to designate a similar loudness relation for pure tones; the three levels are just audible, pleasant, and sharp.

A few words, however, should be said concerning the binaural balance technique. It concerns the sequence of test tone application. It could regularly be demonstrated that the ear offered the test tone last has a relative advantage of loudness perception over the one hearing the tone first. The phenomenon can also be demonstrated in normal ears, and it is therefore necessary to measure the effect in both sequences, i. e., right and left and left and right. The differences, however, are not great.

B. Hood's Perstimulatory Test.—Affiliated with the above-described phenomenon are Hood's [25] adaptation experiments, which ought to be mentioned here. Hood describes the simultaneous balancing technique, in which procedure two ears are balanced first in the normal way and then a simultaneous loudness equalization is pursued in continuous application of the tone at above threshold level for two minutes to both ears. The subject receives a tone of constant intensity at his diseased ear; in my subject it was 80 db. (Fig. 3). At his normal ear the output of the telephone can constantly be brought up and down to match the loudness of the dis-

§ Personal communication to the author.

‖ References 42 and 33.

eased side. As it appears from the diagram (broken lines), the subject constantly turns down the output at the normal side during the first minute to stabilize his equal-loudness impression at both sides. This equalizing effect appears to be unexpectedly large in these cases in which impairment at the diseased side is of the recruitment type. This is seen from the slope of the curves. Whereas originally the slope approached a horizontal level (80 db. at the diseased side and 70 db. at the normal side), after one minute this slope grew so much steeper (80 db. at the diseased side and 30 db. at the normal side) that it even surpassed the slope of the threshold balance (30 db. at the diseased side and 0 db. at the normal side) by 20 db. Thus a negative recruitment of 20 db. takes the place of the original positive recruitment of 20 db.; the effect was described by Hood as recruitment reversion.

This seemed a very interesting phenomenon, and I was anxious to verify Hood's provocative findings. At my disposal were many cases of acoustic trauma. That cochlear damage ensues in these cases is now a well-established fact. The result was negative; I did not find a clear recruitment reversion. Loudness equalization, being once achieved, remained more or less constant during the two following minutes. I say "more or less," because it is hard to assess the loudness matching under these binaural simultaneous test circumstances and even more so because the tones differ in pitch and in timbre (diplacusis and dysacousis). A minor shift in favor of the normal ear is indeed sometimes encountered, but there are no large deviations warranting the name of recruitment reversion. It becomes a ticklish affair to build a diagnostic frame when so many aberrations from normality impinge upon the test. Langenbeck ¶ also found no recruitment reversion in cases of acoustic trauma. Why do my results differ so much from Hood's? Is it because his patients mainly had Ménière's disease, whereas mine were suffering from acoustic trauma? Disturbances in the efferent fibers might possibly play a role. Recent studies by both Portmann [41] and Bocca # have placed the existence of these fibers beyond any doubt.

I feel that Hood's test is a difficult one. When no initial balance is possible at equal intensities, as in Hood's case (Fig. 3) (80 db. at the diseased side and 70 db. at the normal side), then right from the start the diseased side is at a disadvantage because it has to cope with the burden of 10 db. higher sound pressure; consequently, this diseased ear will evince fatigue quicker and to a larger extent than the normal ear, making possible a further decrease of the sound pressure at the normal side. I do not believe that great clinical value can as yet be attached to the test. In the next paragraph I shall enter into this problem a little further.

Reger [85] is investigating a similar phenomenon in a slightly different way when he notices a fast rise in threshold for those tones that show recruitment. The above threshold test by Hood relies on the difficult perstimulatory comparison between both ears, as outlined above, whereas Reger's changing threshold test does not involve the complications inherent to this binaural method, and it can also be applied in cases in which both ears have sustained damage. Reger, still being apprehensive of identifying this phenomenon with fatigue, has named it temporary threshold shift. My results with his test on material consisting to a large extent of cases of acoustic trauma have been rather equivocal. In some 50% of my cases I did not find a temporary threshold shift exceeding 5 db. Reger mentions that he did not find a

¶ Personal communication to the author.

Read before the Fifth International Congress of Oto-Rhino-Laryngo-Broncho-Esophagology, Amsterdam, June 8, 1953.

temporary threshold shift in all cases of recruitment. Would the absence of the temporary threshold shift reflect a nosologic entity as we know it to exist in acoustic trauma?

C. Lüscher's Test.—To conclude this section on recruitment testing technique, my experiences with the difference-limen method described by Lüscher and Zwislocki [26] will shortly be dealt with. As is known, this technique tries to establish the just noticeable fluctuations at 40 db. above threshold. In recruitment cases even very small fluctuations should be noticeable. In my material I could not find by this method any reliable information which was in agreement with the negative results obtained by Lauritz Lund-Iversen.[27] Von Békésy's [28] difference-limen method is principally different because it takes the audiogram at threshold where, as it is known, recruitment usually is at its maximum.

The same objection to testing recruitment so much above threshold was made by Huizing * and by Jerger.[29] Jerger's test is performed at 20 db. above threshold and has been given the name Northwestern Difference Limen test. My negative findings with the Lüscher test did not surprise me very much, because the loudness curve in cochlear impairment takes a course which is usually almost normal at 40 db. above the impaired threshold. In sections IV. B. 4. (Fig. 1) and IV. C. (Fig. 2) I stated that the delayed-recruitment curve was typical of impedance-changing effects. When, on the other hand, the lesion is in the inner ear and, self-evidently, the other ear has no hidden recruitment, a balance curve will not deviate materially from the type given by Steinberg and Gardner,[30] which means normal loudness at 40 db. above threshold.

Denes and Naunton [40] measure the difference limen at both 5 and 45 db. above threshold. In normal and deaf ears without recruitment the difference limen decreases with intensity, whereas the difference limen remains the same or increases when recruitment is present. The complexity of recruitment is, however, not fully covered by Steinberg-Gardner function. How does one account for the overrecruitment? In the next section I shall discuss this remarkable phenomenon.

VIII. COMMENT

A. General Considerations.—The data at my disposal permit me to venture a schematic representation of cochlear mechanisms in both normal and pathological circumstances (Fig. 4). It will be seen that the origin of recruitment is, in my opinion, not identical for the several functional diseases. Whether this provisional and discreet attempt at depicting the cochlea is yet too audacious, I do not know; it helps, however, in correlating facts. Until more is known about the loudness representation in the eighth nerve—the relation between sound pressure and nerve potential—transferring functional facts into anatomical language remains a speculative and haphazard affair. More data like those of Kemp, Coppée, and Robinson [44] are wanted.

It is not the purpose of this paper to submerge into the many eddies and waves of the inner-ear fluids, the neural responses, and the brain that have been hypothesized by the investigators of hearing function. I shall follow Wever's dualistic theory of spatial and temporal representation of pitch and loudness in the nerve response. That, according to Wever, loudness involves the same physical dimensions of space and time as pitch does has met with much opposition. The arguments presented by

* Huizing, H. C.: In discussion on Lüscher and Zwislocki.[26]

Wever are, however, very strong and convincing, the "buzz-and-body" theory of Davis actually acknowledging the same principle. The great divergence of opinion among the "place partisans"—whether it is resonance (Helmholtz, Wever) or the traveling wave (Békésy, Davis) that determines the locus of stimulation—thus becomes of academic interest, since neither hypothesis can account for a sharp tuning at all frequencies and must therefore have recourse to additional factors. As mentioned, Wever acknowledges for this reason frequency as an additional factor in pitch determination, whereas Davis more floridly calls it buzz and body. The magnificent experimental work by both authors can not be further outlined here.[36]

B. Acoustic Trauma (Fig. 4, II).—The gap in the continuity of hair cells known from experimental research to be the corollary of acoustic trauma is totally or pre-

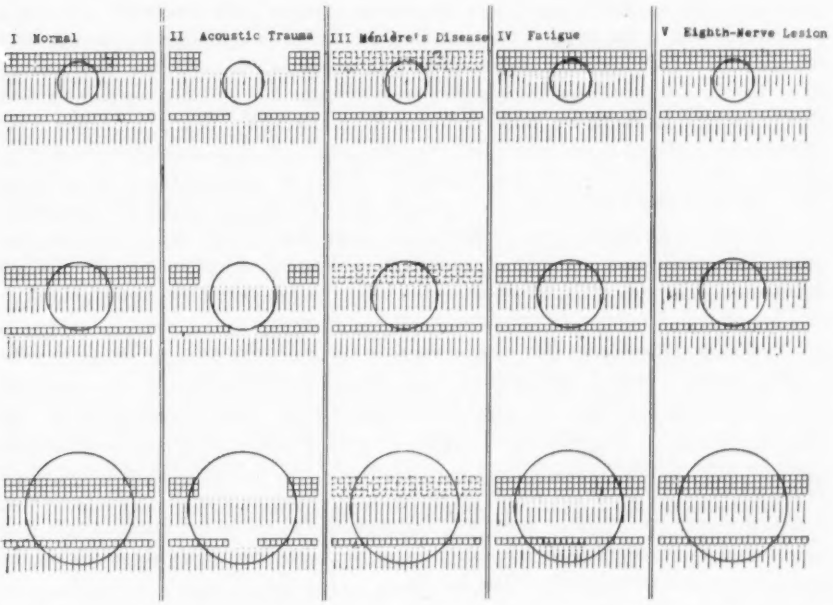


Fig. 4.—Stimulation patterns are visualized in the cochlea under normal and several pathological circumstances. Three outer hair-cell rows, nerve-fiber terminations, nerve fibers, and one inner hair-cell row constitute the picture. Loudness relations are represented at 10 db. in row *A*, at 40 db. in row *B*, and at 70 db. in row *C*.

dominantly confined to the outer rows (Fig. 4, II). The damage to the cochlear structures can clearly be demonstrated in the cochlear microphonics. From the representation in Figure 4, II, it can be seen that no sound impulse can be relayed to the nerve fiber terminations at low sound pressures (*A*), whereas at higher outputs this loss is annulled, because the lack of impulse contribution due to the missing cells diminishes constantly with rise in output, which tallies with the explanation given by Steinberg and Gardner.[30]

The disruption in the hair-cell rows will change the pattern from which dysacousis may result, whereas the anatomical disfigurement of Corti's organ will change the sound waves and cause diplacusis. The extra burden to the normal hair cells

at both ends of the gap now sustaining the full impact of the pressure wave will result in broadening the discharging hair-cell area, thus giving rise to the overrecruitment. The total nerve discharge will perhaps be reduced, but if the region of contributing fibers (whether or not there is a central gap) is enlarged, it will be interpreted as increase in loudness.

C. Ménière's Disease (Labyrinthine Hydrops) (Fig. 4, III).—The basic functional defect will probably be a lesion of the more vulnerable outer hair cells, comparable to the impairment suffered from anoxia, anemia, or other noxious agents. If cochlear microphonics could clinically be taken, they would probably show a reduced output (I hope that the reader will forgive my prediction). As long as the inner hair cells remain normal, they will furnish a loudness impression as great as in normal circumstances or even greater if they are burdened with an extra load when the tectorial membrane misses its normal support from the outer hair cells; overrecruitment is the result. The threshold loss is, of course, due to the fact that the inner hair cells are only starting their excitatory activity at levels much above those that will normally produce a discharge in outer hair-cell fibers. Biochemical changes in the labyrinthine fluids resulting from arteriolar spasms and other vascular crises in the stria vascularis will damage Corti's organ in much the same way, as was already described by van Lennep [31] (1910) in investigations on waltzing mice; his beautiful microscopic photographs show the ectasia of the cochlear duct. Biochemical disturbances,[5] not overpressure, cause the damage, as is so often contended. Overpressure as such is immaterial, although it might slightly change mechanical relations in sound transmission; the relative reduction, if any, through this factor will, however, be the same at all sound pressures.

Experimental evidence that overpressure in the inner ear does not interfere with cochlear activity came from Lempert [18] and Krejci and Bornschein,[32] yet I will not so peremptorily deny any influence from overpressure, because indirectly damage may be sustained: impeded blood supply to and impeded drainage of catabolites from an area of abnormal pressure is conceivable. But it is not the absolute value of the pressure itself but the abnormal pressure gradient between blood or subarachnoidal pressure and cochlear pressure. Damage also to the nerve fibers in this way might lead to an accelerated adaptation of nerve fibers and thus play a role in the recruitment reversion, as described by Hood [25] (Fig. 3).

One might speculate next on the cause of diplacusis in Ménière's disease. The physical properties of the inner-ear structures (Corti's organ) as well as the physical properties of the inner-ear fluids determine the locus of maximum stimulation. This holds as well for the adherents of the traveling-wave theory as for the followers of the resonance principle. Tension in the basillar membrane, elasticity in the tectorial membrane, viscosity of the endolymph, and several more factors might undergo changes as a corollary of labyrinthine hydrops. How critical these physical factors are to the minute pitch discriminatory function of the cochlea may be inferred from several experiments, among which I name the one in which diplacusis can be effected by douching the ear with cold water, as shown by Voogd.[39]

A marked diplacusis is also found after fenestration. I am inclined to ascribe this chiefly to the serous labyrinthitis, resulting in changes similar to those outlined above for labyrinthine hydrops, although the displacement of the fenestra ovalis might also contribute its part. The diplacusis in postfenestrated ears is very pronounced for the

high frequencies and is not found in the lower frequencies, which snugly fits in with Wever's concept that pitch in the lower frequencies is determined by the synchronic volley discharge and not by the locus of maximum stimulation. (I am indebted to Dr. Julius Lempert who enabled me to continue part of these investigations as resident in the Endaural Hospital.)

D. Fatigue (Fig. 4, IV).—In fatigue no loss exists in cochlear microphonics; it may reasonably be assumed that no damage is suffered by the hair cells. The effect must be one of nerve-fiber adaptation, with phase retardation skipping of impulses in the single fibers and equilibration due to reduced excitability in the relative refractory period. At levels above the fatiguing one, the stimulating sound impulse covers an area bordered by unfatigued, fresh nerve fibers, whereas in the center of this area the excitatory stimulus is so large that it will easily overcome the impediment to excitation. The total volley of the nerve impulse will consequently be nearly normal. The action potential spikes in adapted fibers may be of reduced voltage, but they will not preclude the impulse being passed on to the cortex.

E. Eighth-Nerve Lesion (Fig. 4, V).—A diffuse lesion of the eighth nerve will show no recruitment, because the relative reduction in discharge remains the same at all levels. Opinions are still controversial on the functional characteristics of the diseased hearing function. The absence of recruitment found by Hallpike [4] and Williams [33] has been doubted by the Fowlers, father and son.† In five cases of eighth-nerve tumor they did find recruitment. They suggest that faulty conclusions might have been drawn from poor or no masking. The difficulties from this angle were discussed above (section IV. *A*). Reger [35] in a case of eighth-nerve tumor found no recruitment but marked fatigue.

The given concise attempt at schematically explaining the presence or absence of recruitment under various circumstances should not be taken too rigidly; it has been my aim to explain recruitment in terms of Wever's dualistic theory of hearing. I cannot agree therefore with Hood's [25] contention that loudness is essentially proportional to the number of active neural units, since his theory ignores the spatial factor. Can I endorse the other statement postulated by Hood that fatigue is located in the end-organ? Cochlear microphonics do not show the effect; the hair-cell function is unimpaired, but if the nerve endings around the hair cells and not the hair cells proper are meant, the facts seem to corroborate his view.

IX. CONCLUSIONS

1. The recruitment phenomenon has proved its clinical importance in diagnosis and therapy.
   - A. Differential diagnosis in cases of perceptive deafness can usually be based on the absence or presence of recruitment. The functional testing, however, becomes difficult in many cases of partial recruitment in which complex situations exist.
   - B. In the prescription of hearing aids a thorough knowledge of what recruitment does to speech intelligibility is of paramount importance.
2. The recruitment phenomenon has deepened insight into the mechanism of sound perception.

† Fowler, E. P., Sr., and Fowler, E. P., Jr.: In discussion on Reger and Kos.[35]

A. Fatigue, adaptation, and trauma have their repercussions on hearing. Recruitment testing is one of the means to reveal the intricacies of these temporary or permanent hearing impairments.

B. The middle-ear muscles can change the impedance of the sound conduction apparatus: The change varies for the different frequencies and for bone and air conduction and depends on the status in the cavum tympani.

3. Further research is needed to clarify many more aspects of hearing under physiological and pathological circumstances.

A. It should be realized that normal loudness relations are finally represented by two dimensions, space and time.

B. It should also be appreciated that pathological processes in hearing usually involve more than one station in the long road from drum membrane to cortex. Both these factors make any oversimplification in explaining hearing phenomena futile.

Recruitment is an accelerated "loudness sensation" versus "sound pressure" function. Because such an accelerated function can be due to different pathological processes, a further functional differentiation of these pathological processes is wanted. When, finally, I survey and muster the facts collected in this paper, I realize that a wide array of recruitment types exist, necessitating a painstaking assay of the abnormal hearing function as to (1) the course of the accelerated function and (2) the absence or presence of accompanying phenomena with respect to quantitative analysis of these accompanying phenomena, diplacusis, dysacousis, and fatigue.

X. SUMMARY

A survey is given of the modern aspects of recruitment. My investigations are presented on the following:

1. Partial recruitment in conduction deafness
2. Partial recruitment in bone conduction
3. Recruitment in herpes oticus
4. Recruitment in multiple sclerosis
5. Newer testing techniques.

Hood's finding of accelerated perstimulatory fatigue in cases of Ménière's disease found no substantiation in my tests in cases of acoustic trauma. A diagrammatic representation of cochlear relations in both normal and pathological circumstances is tentatively advanced.

REFERENCES

1. Harris, J. D.; Haines, H. L., and Myers, C. K.: Loudness Perception for Pure Tones and for Speech, A. M. A. Arch. Otolaryng. **55**:107, 1952.

2. de Bruïne-Altes, J. C.: The Symptom of Regression in Different Kinds of Deafness, Thesis, Groningen, Holland, 1946.

3. Fowler, E. P., Sr.: Marked Deafened Areas in Normal Ears, Arch. Otolaryng. **8**:151, 1928.

4. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: Observations upon Loudness Recruitment Phenomenon, with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear and 8th Nerve, Proc. Roy. Soc. Med. **41**:516, 1948.

5. Fletcher, H.: Loudness, Masking and Their Relation to the Hearing Process and the Problem of Noise Measurement, J. Acoust. Soc. Am. **9**:275, 1938.

6. Mygind, S. H.: Further Labyrinthine Studies, Acta oto-laryng., Supp. 68, p. 53, 1948.

7. Veckmans, J. I.: Essai d'explication du recruitment, Acta oto-rhino-laryng. belg. **3**:355, 1949.

8. Wever, E. G.: Theory of Hearing, New York, John Wiley & Sons, Inc., 1950.

9. Wever, E. G.; Lawrence, M., and Smith, K. R.: The Middle Ear in Sound Conduction, Arch. Otolaryng. **48**:19, 1948.

10. Wever, E. G., and Lawrence, M.: The Transmission Properties of the Middle Ear, Ann. Otol. Rhin. & Laryng. **59**:5, 1950.

11. Metz, O.: Studies on the Contraction of the Tympanic Muscles as Indicated by Changes in the Impedance of the Ear, Acta oto-laryng. **39**:397, 1951.

12. Kristensen, H. K., and Jepsen, D.: Recruitment in Otoneurological Diagnostics, Acta oto-laryng. **42**:553, 1952.

13. Bornschein, H., and Krejci, F.: Bioelectrische Functionsanalyse der intraauricul Muskulatur, Monatsschr. Ohrenh. **86**:221, 1952.

14. Wever, E. G., and Bray, C. W.: The Stapedius Muscle in Relation to Sound Conduction, J. Exper. Psychol. **31**:35, 1942.

15. Mol, H.: Technische verklaring van het mechanisme van het horen na fenestratie, in Transactions of the Netherlands Laryngological, Rhinological, and Otological Society, Leyden, Nov. 19-20, 1949, Nederl. tijdschr. geneesk. **95**:85, 1951.

16. Mol, H.: Het dynamische gedrag van het oor na fenestratie, in Transactions of the Netherlands Laryngological, Rhinological, and Otological Society, Utrecht, Nov. 25-26, 1950, Nederl. tijdschr. geneesk. **95**:2643, 1951.

17. Mol, H.: The Dynamic Behavior of the Ear After the Fenestration Operation, Acta oto-laryng. **39**:409, 1951.

18. Lempert, J., and others: Perilymph: Its Relation to the Improvement of Hearing Which Follows Fenestration of the Vestibular Labyrinth in Clinical Otosclerosis, Arch. Otolaryng. **50**:377, 1949.

19. Smith, K. R.: Bone Conduction During Experimental Fixation of the Stapes, J. Exper. Psychol. **33**:96, 1943.

20. Groen, J. J.: Medische Physica, Amsterdam, North Holland Printing Company, 1949, p. 592.

21. Carhart, R.: The Clinical Application of Bone Conduction Audiometry, Arch. Otolaryng. **51**:798, 1950.

22. McConnell, F., and Carhart, R.: Influence of Fenestration Surgery on Bone Conduction Measurements, Laryngoscope **62**:1267, 1952.

23. Biemond, A.: Over de zogenaamde Haematorrhachis, Nederl. tijdschr. geneesk. **94**:3145, 1950.

24. von Leden, H., and Horton, B. T.: Auditory Nerve in Multiple Sclerosis, Arch. Otolaryng. **48**:51, 1948.

25. Hood, J. D.: Studies in Auditory Fatigue and Adaptation, Acta oto-laryng., Supp. 92, p. 1, 1950.

26. Lüscher, E., and Zwislocki, J.: A Simple Method for Indirect Monaural Determination of the Recruitment Phenomenon (Difference Limen in Intensity in Different Types of Deafness), Acta oto-laryng., Supp. 78, p. 156, 1948.

27. Lund-Iversen, L.: An Investigation on the Difference Limen Determined by the Method of Lüscher-Zwislocki in Normal Hearing and in Various Forms of Deafness, Acta oto-laryng. **42**:219, 1952.

28. von Békésy, G.: A New Type Audiometer, Acta oto-laryng. **35**:411, 1947.

29. Jerger, J. F.: A Difference Limen Recruitment Test and Its Diagnostic Significance, Laryngoscope **62**:1316, 1952.

30. Steinberg, J. C., and Gardner, M. B.: The Dependence of Hearing Impairment on Sound Intensity, J. Acous. Soc. Am. **9**:11, 1937.

31. van Lennep, E. C. C.: Abnormalities in the Hearing Apparatus Demonstrated in Waltzing Mice, Thesis, Utrecht, Holland, 1910.

32. Krejci, F., and Bornschein, H.: Tierexperimentelle Untersuchungen über die Cochlearfunktion bei endokranieller Drucksteigerung, Pract. oto-rhino-laryng. **13**:146, 1951.

33. Eby, L. G., and Williams, H. L.: Recruitment of Loudness in Differential Diagnosis of End-Organ and Nerve Fiber Deafness, Laryngoscope **61**:400, 1951.

34. Lempert, J., and others: New Theory for the Correlation of the Pathology and Symptomatology of Ménière Disease, Ann. Otol. Rhin. & Laryng. **61**:717, 1952.

35. Reger, S. N., and Kos, C. M.: Clinical Measurements and Implications of Recruitment, Ann. Otol. Rhin. & Laryng. **61**:810, 1952.

36. Wever, E G., and Lawrence, M.: Sound Conduction in the Cochlea, Ann. Otol. Rhin. & Laryng. **61**:824, 1952.

37. Huizing, H. C.: The Relation Between Auditory Fatigue and Recruitment, Acta otolaryng., Supp. 78, p. 169, 1948.

38. van Dishoeck, H. A. E.: De diagnose van catarrhale middenoor ontstekingen, Nederl. tijdschr. geneesk. **97**:1306, 1953.

39. Voogd, J.: Ned. Keel. Oor. Ver., 1946.

40. Denes, P., and Naunton, R. F.: The Clinical Detection of Auditory Recruitment, J. Laryng. & Otol. **64**:375, 1950.

41. Portmann, G.; Portmann, M., and Portmann, C.: La double innervation de l'organe de Corti, Acta oto-laryng. **43**:226, 1953.

42. Huizing, H. C., and Reyntjes, J. A.: Proceedings of the Collegium Oto-Rhino-Laryngologicum Amictiae Sacrum, 1950.

43. Lansberg, M. P.: Moelijkheden bij de indicetrestelling tot fenestratie operatie, Nederl. tijdschr. geneesk. **92**:3590, 1944.

44. Kemp, E. H.; Coppée, G. E., and Robinson, E. H.: Electric Responses of the Brain Stem to Unilateral Auditory Stimulation, Am. J. Physiol. **120**:304, 1937.

CIRCULATION OF THE SPIRAL LIGAMENT AND STRIA VASCULARIS OF LIVING GUINEA PIG

Experimental Study

FRANCIS L. WEILLE, M.D.
SAMUEL R. GARGANO, B.S.
RAYMOND PFISTER, M.D.
DANIEL MARTINEZ, M.D.
AND
JOHN W. IRWIN, M.D.
BOSTON

THE POSSIBILITY of studying the blood vessels of both the spiral ligament and stria vascularis of living guinea pigs occurred to one of us (F. L. W.). The purposes of this article are to describe the exposure of the spiral ligament and stria vascularis in living anesthetized guinea pigs and to present a series of microscopic observations on the small blood vessels of this area.

During the past century both histologists and otologists have been intensely interested in the circulation of the inner ear. Using an array of various histological techniques, many investigators have attempted to define with accuracy the small blood vessels of the spiral ligament and stria vascularis (for instance, Huschke,[1] Retzius,[2] Voltolini,[3] Bœttcher,[4] Schwalbe,[5] Siebenmann,[6] Shambaugh,[7] Nabeya,[8] Belemer,[9] Agazzi,[10] and Smith [11]).

Certain basic differences of opinion have arisen involving the morphology and physiology of these vessels. Shambaugh [7] found three capillary networks in the cochlea of pigs, whereas Smith [11] described four capillary networks in the spiral ligament and stria vascularis of the guinea pig. Belemer [9] reported that arterioles both fed and drained the capillary network of the stria vascularis. Agazzi [10] demonstrated arteriovenous arcades in the spiral ligament, and he attempted to show that these arcades were important in regulating blood flow in this area.

METHOD

More than 500 healthy guinea pigs, free of vermin and weighing between 200 and 1,000 gm., were the experimental animals. Initially, each animal was injected intraperitoneally with 45 mg. of sodium pentobarbital per kilogram of body weight, and further doses were administered as required during the course of the experiment.

The operative procedure was divided into two distinct parts: the macroscopic exposure of the cochlea and the microscopic fenestration of either the apical or the third cochlear turn. With a scalpel an incision approximately 1 cm. in length was made parallel to the inferior border of

---

Read before the Otosclerosis Study Group, Chicago, October, 1953.

This work was aided by a grant from the William S. Eaton Fund.

From the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary and the Allergy Laboratory of the Massachusetts General Hospital.

the external auditory meatus; then, with a hemostat the skin and subcutaneous tissues were separated from the underlying muscle. Next, the incision was extended, first, anteriorly to just above the eye and then posteriorly to the superior posterior attachment of the pinna. The musculus auricularis anterior and the musculus auricularis posterior were separated with blunt dissection from their pinnal attachments, and the posterior auricular artery was tied and severed. To visualize the bulla, the overlying connective tissue and the attachment of the sternocleidomastoid muscle were removed. A second incision, perpendicular to the first and extending to the inferior border of the mandible, exposed the masseter muscle, which was severed in its midportion. The cartilaginous external auditory canal was opened, and the pinna was separated from its attachment to the wall of the external auditory canal. Finally, the removal of the tympanic membrane, the lateral wall of the bulla, as well as the incus and malleus exposed the cochlea. The gross surgery was then completed, and the guinea pig was placed on the experimental table so that the fenestra of the cochlea could be made with the aid of a microscope.

According to Peck and Hoerr,[12] all mechanical vibrations interfere with accurate microscopic observations. The experimental table, therefore, was constructed to eliminate as many vibra-

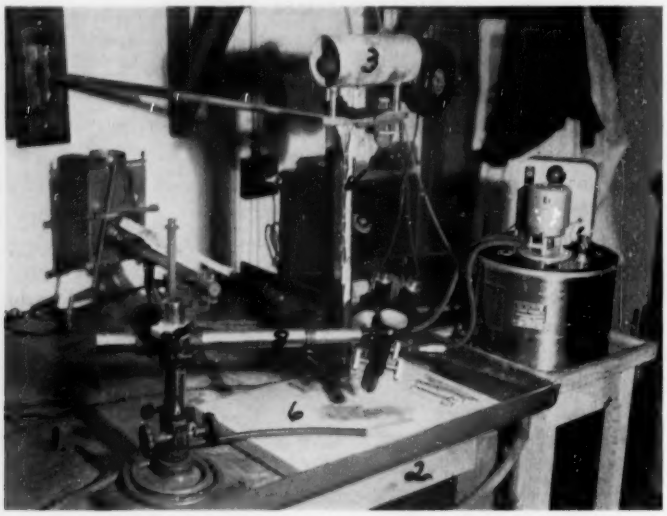


Fig. 1.—Experimental equipment: slab of boiler plate (*1*); steel table (*2*); projection bulb and housing (*3*); fan and adjustable arm (*4*); fused quartz rod (*5*); quartz rod stand (*6*); constant temperature bath (*7*); microscope and microscope's stand (*8*).

tions as possible. This table consisted of a 4 ft. (122 cm.) square, ⅜ in. (1 cm.) thick slab of boiler plate, the four corners of which rested on four pyramids of squash balls held together in steel cups, a heavy steel table with an adjustable top on which the cups rested, and rubber shock absorbers (of the type used for mounting automobile engines) which supported the heavy steel table.

To transilluminate the cochlea a General Electric T-12 1,000 watt projection bulb was the light source. Air from a fan attached by an adjustable arm to the wall of the room cooled this lamp. The light was conducted from the bulb to the cochlea by a fused quartz rod similar to that described by Hoerr.[13] The quartz rod and projection bulb were mounted on a stand which contained a housing for the lamp, held the quartz rod rigid, and at the same time permitted controlled movements of the rod in a vertical as well as in a horizontal plane. Warm (38 C.) mammalian Ringer's solution flowed gently from the hollow tip of the quartz rod at the rate of 3 to 4 cc. per minute. A constant temperature bath and circulating system mounted on a separate table maintained the stream of Ringer's solution at about 38 C. Figure 1 shows the experimental equipment.

For fenestration and microscopic observations, either a Leitz stereoscopic microscope (× 48 to × 150) or a Leitz monobjective, monocular compound microscope (up to × 200) mounted on a stand with an adjustable arm was used. The microscope was focused on the cochlea at an area where the pigment cells of the stria vascularis showed through the bony capsule of the cochlea, and with a curette the bone over the third, or apical, cochlear turn was carefully removed so as not to injure the underlying spiral ligament. The minimum size of such a fenestra was 0.25 by 0.1 mm.

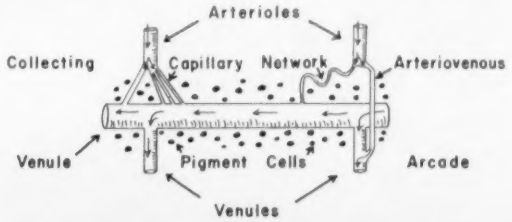


Fig. 2.—Diagram of blood vessels in the area of the spiral ligament.

Fig. 3.—A branching arteriole (*1*) and pigment cells (*2*).

On occasion, in order to dispense with respiratory movements which interfered with microscopic observations made at × 150 to × 200, a tracheotomy was performed. Then a tracheal cannula which only filled two-thirds of the tracheal lumen was inserted. When oxygen under sufficient pressure was delivered through this cannula, respiratory movements ceased.

OBSERVATIONS

On focusing down on the spiral ligament through the fenestra with the microscope (× 60), one was impressed first by the narrow band of pigment cells in the middle of the field. Toward the apex of the cochlea one could see one to three blood vessels which branched as they approached the base. Some of these branches appeared to sink in among the pigment cells; others ran over the pigment cells to join vessels running perpendicular or parallel to the pigmented area, and still others joined vessels running through the pigmented-cell area.

Fig. 4.—Arteriovenous arcades (*1*) and a collecting venule (*2*).

Fig. 5.—An arteriole (*1*); capillaries of the spiral ligament (*2*); pigment cells (*3*).

On more careful examination at magnifications up to × 200, it was possible to study these vessels morphologically. In order to familiarize the reader with the blood vessels of the area under observation, Figure 2 is presented. The blood vessels which ran from the upper part of the fenestra toward the base of the cochlea were

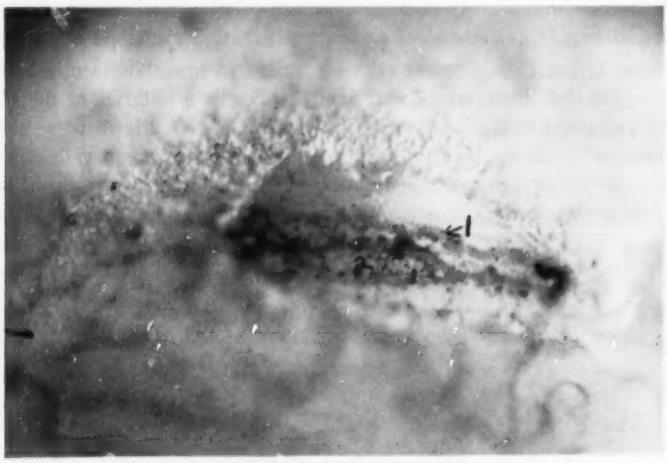


Fig. 6.—Collecting venules (*1*) and pigment cells (*2*).

Fig. 7.—A collecting venule (*1*) and a venule (*2*).

called arterioles, as they took the shape of cones with the apex of the cone toward the cochlear base and the direction of linear blood flow was to the branches. Some branches were given off to a capillary network in the spiral ligament; other branches sank deep among the pigment cells to feed the capillary network of the stria vascularis; finally, a few branches passed directly over the area of the stria vascularis

and entered venules. These last branches were no doubt the same ones which Agazzi [10] called arteriovenous arcades. Figure 3 visualizes a branching arteriole, and Figure 4 shows an arteriovenous arcade.

Some branches of the arterioles fed capillaries found in the spiral ligament. These capillaries formed a network of branching and anastomosing cylindrical vessels, which were easy to visualize, as they were near the surface. Venules in the pigmented area drained this network of capillaries. Figure 5 visualizes these capillaries.

In a layer beneath the pigment cells a network of branching and anastomosing cylinders was found. These vessels appeared to be the capillaries of the stria vascularis. The pigment cells often interfered with accurate study of these capillaries, and on occasion it was necessary to remove some of the pigment cells. Since these vessels were in deeper tissue, adequate microscopic resolution was a problem. These capillaries drained into the collecting venules in the area.

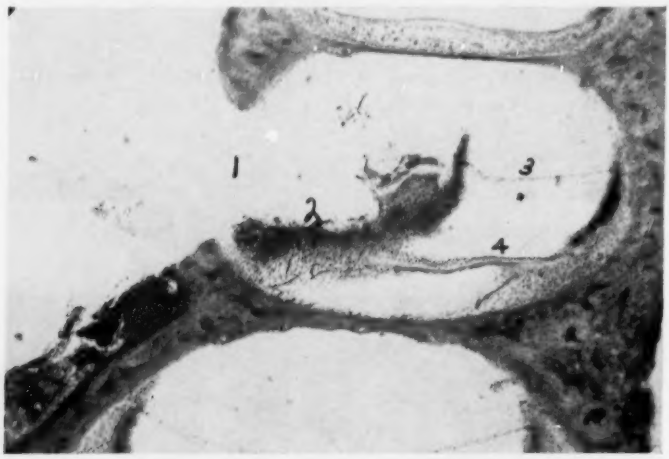


Fig. 8.—Photomicrograph of a section showing the fenestra in the third cochlear turn (*1*); the spiral ligament and stria vascularis (*2*); Reissner's membrane (*3*), and the basilar membrane (*4*).

Two sets of venules were found among the pigmented cells. Those venules which flowed horizontally to the field might be called collecting venules. The direction of blood flow was from the tributaries to the larger vessels. These venules, in turn, flowed into other venules which were perpendicular to them and in the area caudal to the stria vascularis. In Figure 6 the collecting venules are visualized, and in Figure 7 the other venules are seen.

All the arterioles, arteriovenous arcades, and venules were observed to contract and dilate independently. The lumen of each vessel type was seen to disappear completely, only to reappear later. The diameters of certain segments of each vessel were measured repeatedly, and there were significant differences. The question of independent contractility of the two capillary networks was not definitely determined. Single capillaries as well as whole networks were seen to disappear, only to reappear. Such action could be due either to passive collapse because of arterioles closing off or to active contraction.

Rate of blood flow varied in each vessel from time to time. Rates varied from very rapid to no flow at all. Then intermittence of linear blood flow was noted in each type of blood vessel.

COMMENT

An experimental technique has been outlined for studying the small blood vessels of the spiral ligament and stria vascularis of living guinea pigs. As a check on our observations the cochlea of a living guinea pig was fenestrated by the method described; the living vessels of the spiral ligament were visualized; the spiral ligament was fixed in vivo, and then the temporal bone was removed, decalcified, stained, and sectioned. Sections clearly showed that the fenestra was over the spiral ligament and stria vascularis. Figure 8 is a photomicrograph of such a section.

Certain objections to this method might be considered. First, it was necessary to anesthetize the animals, and all anesthetic agents disturb normal physiological relationships. Secondly, on occasion insufflation of 100% oxygen was used to stop respiratory movements. Kety and Schmidt [14] have shown that cerebral blood flow is diminished about 13% by insufflation of 100% oxygen, and it might be true in this instance. Thirdly, considerable surgery was required, and trauma can damage small vessels. Fourth, one was limited to viewing only the vessels on or near the surface. In spite of these obstacles, the circulation in the spiral ligament was studied in some animals for many hours without dramatic pathological changes becoming evident.

SUMMARY

A method for direct microscopic observation of the blood vessels of the important areas of the spiral ligament in the living animal has been described.

Arterioles, capillaries, arteriovenous arcades, and venules of the spiral ligament and capillaries of the stria vascularis were observed.

The arterioles, arteriovenous arcades, and venules possessed the ability to contract and dilate independently.

Intermittence of linear blood flow was observed in arterioles, arteriovenous arcades, capillaries, and venules.

Rate of blood flow varied from time to time in each type of vessel.

247 Commonwealth Ave.

REFERENCES

1. Huschke, R.: Über die Gehörzähne, Mueller's Arch., pp. 335-346, 1835.

2. Retzius, G.: Das Gehörorgan der Wirbelthiere: Morphologisch-biologische Studien, Stockholm, Samson & Wallin, 1844.

3. Voltolini: Über die Gehörzähne der Schnecke des Menschen und der Säugethiere und deren Gefässe, Arch. path. Anat. **104**:109-117, 1886.

4. Bœttcher, A.: Rückblicke auf die neueren Untersuchungen über den Bau der Schnecke, im Anschluss an eigene Beobachtungen, Arch. Ohrenh. **24**:95-166, 1886-1887.

5. Schwalbe, G.: Ein Beitrag zur Kenntniss der Circulationsverhältnisse in der Gehörschnecke, Beitr. Physiol., pp. 200-220, 1887.

6. Siebenmann, F.: Die Blutgefässe im Labyrinth des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1894.

7. Shambaugh, G. E.: The Distribution of Blood-Vessels in the Labyrinth of the Ear of the Sus Scrofa Domesticus, Chicago, University of Chicago Press, 1903, Vol. 10.

8. Nabeya, D.: A Study in the Comparative Anatomy of the Blood-Vascular System of the Internal Ear in Mammalia and in Homo (Japanese), Acta scholae med. univ. Kioto **6**:1-132, 1923.

9. Belemer, J. J.: The Vessels of the Stria Vascularis, with Special Reference to Their Function, Arch. Otolaryng. **23**:93-97, 1936.

10. Agazzi, C.: Appunti di idrodinamica vascolare del legamento spirale, Arch. ital. otol. **60**:40-47, 1949.

11. Smith, C. A.: Capillary Areas of the Cochlea in the Guinea Pig, Laryngoscope **61**:1073-1095, 1951.

12. Peck, H. M., and Hoerr, N. L.: Intermediary Circulation in the Red Pulp of Mouse Spleen, Anat. Rec. **109**:447-477, 1951.

13. Hoerr, N. L.: Microscopy: Illustration of Living Tissues, in Medical Physics, edited by O. Glasser, Chicago, Year Book Publishers, Inc., 1950, pp. 533-534.

14. Kety, S. S., and Schmidt, C. F.: The Effects of Altered Arterial Tensions of Carbon Dioxide and Oxygen on Cerebral Blood Flow and Cerebral Oxygen Consumption of Normal Young Men, J. Clin. Invest. **27**:484-492, 1948.

RHINOPLASTIC APPLICATIONS TO SUBMUCOUS RESECTION OF THE NASAL SEPTUM

Surgical Anatomy; Technique of a Modified Procedure

ALAN L. ROWLAND, M.D.
SAN DIEGO, CALIF.

SINCE submucous resection of the nasal septum is probably the most frequently performed operation still wholly in the domain of the otolaryngologist, in the light of the many recent studies on the physiologic functions of the nose, and in view of the many developments which have recently been forthcoming on the subject of rhinoplasty as a restorative process for the amelioration of disturbed nasal mechanism, it is felt that it may be beneficial to reflect again on the rationale of the operation and to offer a suggestion for its possible improvement in the light of these studies. I believe that there is merit in a reappraisal of the traditional operation and in attempts toward improvement of a technique which has been so fixed that it is rare to find a paper on submucous resection in the literature of recent years.

As in any other operative procedure on the nose, an understanding knowledge of the surgical anatomy of the septum is necessary in order to apply intelligent reasoning in carrying out surgical applications for the correction of deviation of the nasal septum. The upper lateral cartilages are continuous with the cartilaginous septum at their anterior borders through their upper portion, so that they form in reality only one cartilage, whose form is that of a central theoretically straight lamina with two firmly attached, contiguous lateral wings. Hence in any deviation or twisting of the middle cartilaginous vault, the septum is invariably involved. However, toward the nasal tip there is a complete separation of the anterior borders of these cartilages. In their inferior portion they are completely separated from the septal cartilage, at this point forming a dehiscence, which results in a depression of the dorsum of the nose in this area. Here the line of the dorsum is supported only by the anterior border of the septal cartilage, a fact which here explains the weakness of the dorsum and which results in the increased frequency of depressed dorsal lines in their midportions (Fig. 1). A typical example of this, which is often observed, is that of "boxer's nose," in which the nose is subjected to frequent and recurring trauma in its entirety but in which the bony vault is straight, as is the lower cartilaginous vault, and the middle cartilaginous vault is almost invariably depressed or deviated. Because of the extremely intimate connection between the upper lateral cartilages and the septum, the septal cartilage is invariably involved in middorsal deviations, and the latter cannot be corrected without a concomitant submucous resection of the septum itself.

The cartilage of the septum also provides the basic supportive framework for the base of the nose (Fig. 2). Its anterocaudal border rests between and in the closest proximity with the mesial crura of the lower lateral cartilages of the nose. Thus there is no deformity of the columella (which consists chiefly of these mesial crura)

in which the septum is not involved. Twisted tip, deflected tip, retracted tip, hanging columella—these are all conditions which cannot be corrected without the accompanying correction of the deviated septum in its lower portion.

In dislocation of the septum, a condition commonly observed, the dorsal line of the nose is often displaced in its lower third, since the septum here also forms the large part of the supportive framework externally. This dislocation therefore results

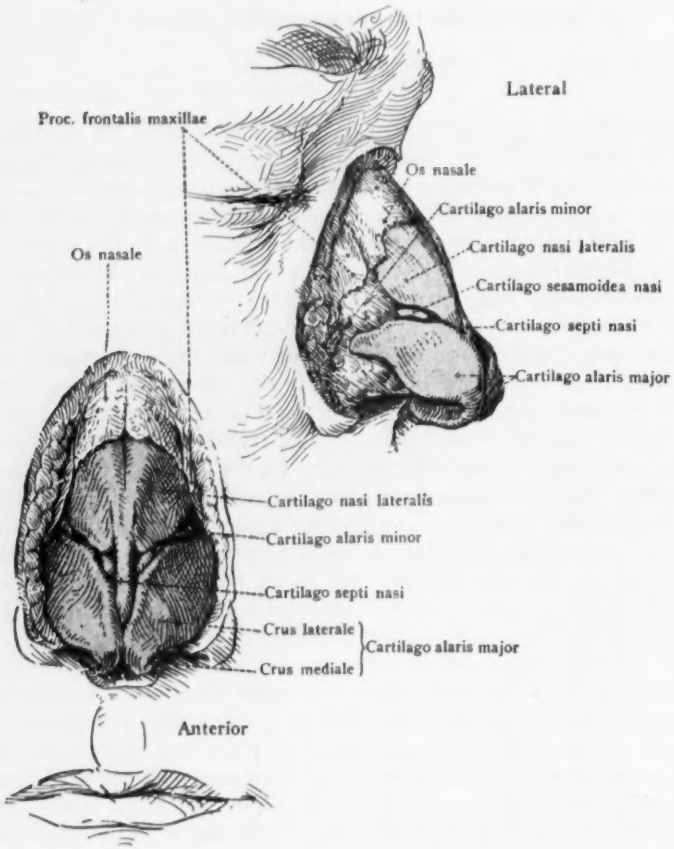


Fig. 1.—Anterior and lateral view of cartilages of nose.

at the same time in a displacement of the adjacent part of the dorsal arch in its lower third, to which it is intimately in relation and which cannot be corrected without appropriate straightening of the displaced septum.

Thus it is seen that the septal cartilage is involved in almost every nasal disfigurement. Even in deviations of the bony septum, in which, owing to the rigidity and relative stability of the overlying nasal bones, one would expect a minimum of concomitant deflection of the lower cartilaginous, more malleable portions, it is actu-

ally observed that in these conditions the bony deviation carries with it the lower cartilaginous and anterior vomerine elements because of the extremely intimate connection of these elements at their common fibrous points of junction.

The operation of submucous resection of the nasal septum has been done for many years in a routine manner. Killian introduced his incision years before nasal physiology was well known. Although his operation has stood the test of time relatively well, is it still not the moment to try to adapt the procedure to the newer concepts introduced by the many studies on physiologic function and to attempt to correlate it to the numerous recent developments in rhinoplastic repair?

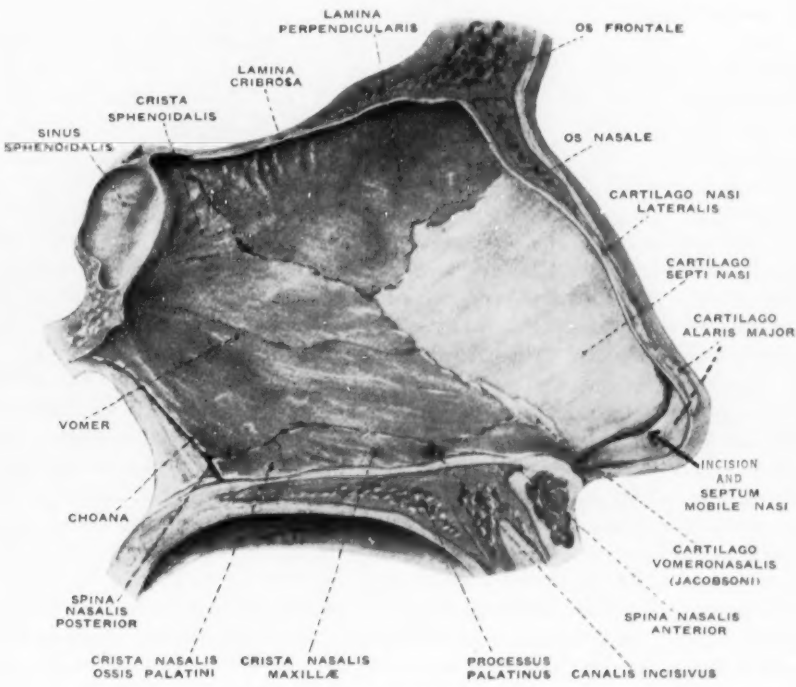


Fig. 2.—The septum nasi (Loeb).

A deviation of the external cartilaginous nose may be secondary to a deflection of the bony nose or to that of the nasal septum, as has already been seen. A deviation of the lower two-thirds of the external nose, whatever its cause, cannot be corrected in the presence of a markedly deviated septum, and it is often unamenable to correction in the presence of any but the most minor deviations of the septum. Among the results of such a deviation, translated to the external nose, are the deflected tip, twisted tip, and most columellar deformities. The surgical anatomic causes for these malformations have already been discussed. Therefore it has seemed to me that submucous resection done according to rhinoplastic principles will more successfully correct these allied conditions than the heretofore traditional operation and that the former conforms more closely at the same time to the basic new dictum of disturbing as little as possible the normal physiologic structure and function of the nasal

mucosa.[1] That the classic incision of Killian, to say nothing of the higher incisions, involves the integrity of the nasal mucosa is incontrovertible. Jones, in 1942, advocated an incision of the cutaneous side of the mucocutaneous border, as had Colver before. He advocated it because "injury to the mucous membrane many times leaves a scar that causes bleeding and crusting as is seen in old scarred areas without operation; by cutting through the skin side one avoids this after-crusting areas."[2] It will be seen in Figure 2 that in making the Killian-type incision into the nasal mucosa on one side or the other of the nasal septum one cannot avoid incision into the respiratory epithelium, at least through some of its extent. A careful study of Figure 2 will also make plain that there is only one area in which the respiratory epithelium will not be injured and which is at the same time the most logical anatomically. This is a line following the caudal end of the cartilaginous septum and immediately adjacent beneath it. This is the incision made by rhinoplastic surgeons for the repair of malformations of the lower cartilaginous vault and called the transfixion incision.

The operation using this incision and my technique follows. The patient is given 3 grains (0.19 gm.) of pentobarbital (Nembutal) about an hour before operation. The nose is anesthetized by a preliminary packing of cotton or gauze with tetracaine (Pontocaine) hydrochloride, 2%, or equal parts of cocaine, 10%, and epinephrine, 1:1,000, and sparingly applied intranasally in contact with the branches of the anterior ethmoidal nerve. In a few minutes infiltration is done employing procaine hydrochloride, 2%, to which has been added a few drops of epinephrine, 1:1,000. About 0.5 cc. is injected into the points of entry into the nose of the branches of the anterior ethmoidal nerve on either side. Additional sparing infiltration is made at the intraorbital foramen and into the nasopalatine nerve at its point of exit at the maxillary spine on either side of the base of the septum. In addition, minimal infiltration is done of the nose. Incision is made through the septal wall at its base, using a No. 15 blade. It is of the utmost importance that the columella be retracted caudally with the fingers of the left hand and that the mobile or membranous septum be put on the stretch in order to preserve it intact. This retraction may also be done with a special forceps for columellar retraction devised by Cottle. Since the mobile septum has marked contractile properties, this is easily accomplished. The blade is introduced at the anterocaudal extremity of the septum and is made to incise the skin of the vestibule, the membranous septum, and the skin of the opposite nares as close beneath the cartilage of the septum as possible. The incision is then carried dorsally along and hugging the caudal border of the septum through its ventral two-thirds while caudal traction on the columella is maintained (Fig. 2). The incision is completed, Mayo scissors being used to separate the posterior fibrous third of the septum from its attachment to the nasal spine. There is thus in plain view, at the conclusion of this transfixion of the septum, the entire caudal border of the septum, with the mucosal sheaths plainly seen on either side of the cartilage. Separation of the mucous membrane from the cartilage on either side is very easily accomplished, with a minimum of trauma, by blunt dissection or merely by the introduction of the Mackenty elevator. The mucosa is separated or stripped from the cartilage and bone with greater ease than is possible by the use of any other method.

It will be seen that exposure of the entire base of the septum is greater by using the transfixion incision than by the use of the Killian-type incision. The cartilage is easily visualized and, if it is dislocated, may easily be resected and replaced in the midline.

Access to the bony portion of the septum is simpler by this method, and, in the case of a bony spur or of a bony septal deviation, a better view is thus obtained.

If the lower cartilaginous portion is but moderately displaced, it may be sufficient to straighten this portion by merely cross hatching the cartilage itself in order to reduce its resilience and to break its "spring," according to the technique first advocated by Metzenbaum. If this it not sufficient, any further remaining deviation may then be corrected by using scissors, the scalpel, the swivel knife, or the rongeur, depending on the site of the deviation and the consistency of the involved structure.

If the deviation also involves the tip of the nose, the medial portion of the transfixion incision already made is extended by continuing it between the upper and lower cartilages, as in the typical rhinoplasty and in the ordinary manner. After this, the lower rim incision is made between the rim of the vestibule and the lower border of the lower alar cartilage and the lower lateral delivered in the customary manner. The lateral crus may then be molded according to Fomon's technique or shaped and trimmed according to that of Joseph.

If it is desired to insert a cartilaginous batten, this may be cut and molded from that which has previously been removed in correcting the septal deviation, and this may then be inserted at the nasal tip.

After completion of the correction of the septal deflection, approximation of the lips of the incision is done by means of a fine silk suture on an atraumatic needle. Although good approximation is necessary, usually one suture on either side is all that is necessary. Since the approximation is done in an area which is extremely easily seen, this presents no difficulty.

This results in excellent union, and ordinarily within a few days healing has taken place by first intention. After a week has elapsed, the scar is barely visible on either side.

Since there has been a minimum of trauma, very little packing is ordinarily required. I have found it sufficient to place a small fragment of petrolatum (Vaseline) gauze in contact with the incision.

SUMMARY AND CONCLUSIONS

A method of submucous resection of the nasal septum employing rhinoplastic principles is presented. It is believed that this method is physiologically sounder and more functionally correct than that traditionally advocated in that its comparatively greater ease of execution is much less prone to result in encrusting perforations and its greater exposure in less trauma, and that it will preserve more intactly the physiologically functioning mucosa.

625 Broadway (1).

REFERENCES

1. Proetz, A. W.: Essays on the Applied Physioliogy of the Nose, St. Louis, Annals Publishing Company, 1941.

2. Jones, A. C.: Tr. Am. Laryng. Rhin. & Otol. Soc., pp. 331-332, 1942.

Clinical Notes, New Instruments, and Techniques

ADENOIDECTOMY WITH HEMOSTASIS

MAX UNGER, M.D.
NEW YORK

The operation for the removal of the nasopharyngeal tonsil, more frequently called the adenoids, as practiced today, is one of the simplest, easiest, and quickest operations in the whole field of surgery. The most frequently used—in fact, practically the only—instrument is the LaForce adenotome. This consists essentially of a metal box that is open in the rear to fit over the adenoids, with two grooves on the sides of the opening in which a sharp, flexible, ribbon-shaped blade travels like a guillotine to cut off the adenoid tissue caught in the box. A less frequently used instrument is the ring curette. This consists of a handle, a shaft, and a triangular, square, or oblong frame that has a sharp, rigid blade at the end. It is used with a free-hand motion and requires more dexterity to use than the LaForce instrument but can take off a larger amount of adenoid tissue.

If simplicity, ease, and speed were the only criteria by which to judge an operation, these instruments leave nothing to be desired. Unfortunately, however, the use of these instruments is accompanied by a profuse, immediate hemorrhage and a tendency toward postoperative hemorrhage. This is due to the fact that they have sharp cutting edges which leave smooth, clean-cut wounds. As is well known, smooth, clean-cut wounds bleed more freely than crushed and lacerated wounds.

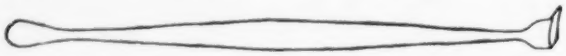


Fig. 1.—Tonsil separator and palate retractor.

Fig. 2.—Adenoid pressure roller.

The operation that I have devised leaves the stump of the adenoid crushed and lacerated so that the amount of immediate hemorrhage is reduced by more than 75% and the tendency toward postoperative bleeding is almost obliterated. To accomplish this purpose, I have devised three instruments.

Figure 1 shows a combined tonsil separator and palate retractor. The separator end is flattened and has fine serrations along its edge, to facilitate dissection of the tonsil and at the same time leave a lacerated stump. The palate retractor end is like a solid-bladed wound retractor and is used to lift up the soft palate and expose the nasopharynx. If the head of the patient is extended and the palate is lifted with the retractor, the entire nasopharynx and the adenoid growth, except a small portion next to the nasal septum, can be seen. This is illustrated in the diagram in Figure 4.

Figure 2 shows my adenoid pressure roller, which I have described elswhere.[1] The essential feature of this is a rough, nobby roller which is rolled over the adenoid base after the adenoid has been removed, and it serves to crush and lacerate the tissues.

Figure 3 shows the new type of adenoid-sawing curette that I have devised, combined with a suction apparatus. As can be seen in the diagram, the handle (*1*), and shaft (*2*), and the ring (*3*) resemble in shape a conventional ring curette. The blade (*4*), however, is basically different. Instead of having a sharp cutting edge, it has an edge that is coarsely serrated so that it becomes a saw, and it is used like a saw, as I shall show presently. Built onto the curette and made integral with it is the suction apparatus, of which *5* is the hose end for attachment of the suction hose, *6* is the main tube which branches at the front end into two tubes (*7*, *7*,), which are closely adapted to the tines of the curette, their terminal ends coming up to the saw

blade of the curette. The suction hose is attached to its own suction pump, or it can be attached to a three-way valve with the suction hose that is used for the throat and tonsil suction tip. A twist of the valve handle can start either instrument to function.

The technique that I use for adenoidectomy is as follows: After I have removed the tonsils by the extrusion-and-suture method that I have described in another paper,[2] the patient's head is held firmly in the midline and slightly extended by the anesthetist or another assistant. The palate retractor is inserted and held by one hand, so that the field of operation is exposed and kept in view throughout the operation. The adenoid sawing curette is now inserted so that the

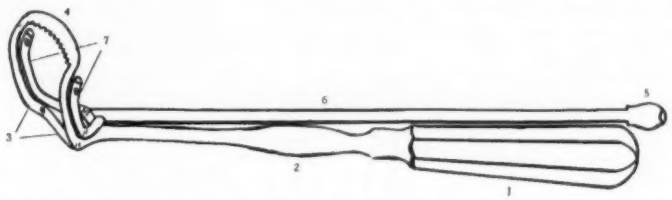


Fig. 3.—Combination adenoid saw curette and suction apparatus: *1*, handle; *2*, shaft; *3*, sides of frame; *4*, saw-edged blade; *5*, hose end of suction tube; *6*, main suction tube; *7,7*, terminal branches of suction tube.

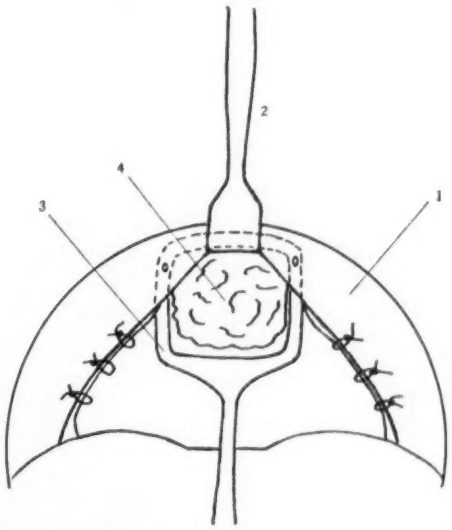


Fig. 4.—Soft palate (*1*) lifted by retractor (*2*). Adenoid curette (*3*) is in position, with adenoid growth (*4*) protruding through frame of the curette.

distal end lies flat against the vault of the pharynx, right up against the posterior edge of the septum, the shaft being in the midline and depressed, so that it is in contact with the lower jaw. The adenoid growth can now be seen protruding through the frame of the curette (Fig. 4). The saw blade is kept in constant contact with the roof of the pharynx and is pushed back against the adenoid growth while at the same time strong short jerks from side to side are given to it. This is illustrated in the diagram in Figure 5. This causes the teeth to saw through the base of the adenoid. This is kept up till the posterior wall of the pharynx is reached. Then the motion of the curette is directed downward. The side-to-side, sawing motion is continued until the entire adenoid growth is amputated. The bleeding that takes place while this is being done is being continuously removed by the suction tubes of the instrument, so that a good view

is possible throughout the operation. As the lower end of the adenoid growth is reached, there may not be enough resistance to the saw blade for the teeth to catch, and they may slip and drag the whole growth down. This is overcome by pressing the palate retractor back against the adenoid so that it is held firmly against the posterior pharyngeal wall above the curette. The lower end of the adenoid is thus converted into a loop, and the teeth of the curette can cut through it. When the adenoid is free, it is held by the suction and can be lifted out when the curette is removed. After the curette is taken out, the regular throat suction tip is put in operation.

The adenoid pressure roller next is introduced up to the front of the roof of the pharynx and rolled with pressure backward and downward over the stump of the adenoid, first in the midline, than on either side. After this, a gauze sponge in a sponge holder is introduced behind

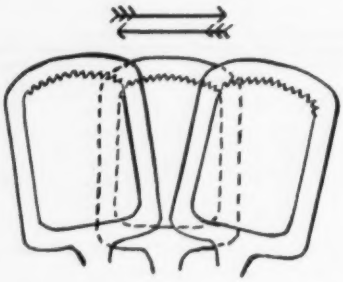


Fig. 5.—Diagrammatic representation of side-to-side excursion of curette that is employed while sawing through base of the adenoid.

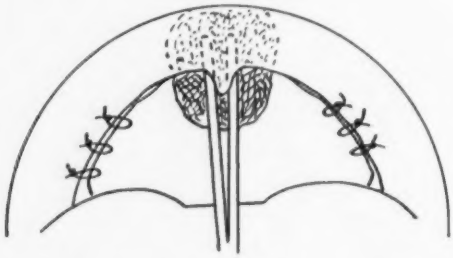


Fig. 6.—Gauze sponge inserted behind the soft palate and pressed against site of amputated adenoid.

the palate and held firmly against the raw tissue for a minute or two (Fig. 6). After this is removed, the field is examined visually, with the help of the palate retractor, for adenoid shreds and bleeding points. If shreds are found, they are caught with forceps and snipped off with scissors or snare. If bleeding points are seen, another application of the roller or gauze sponge is used. The patient is kept on the table till no more bleeding can be detected visually.

As can be inferred from the description of the technique, this operation is not characterized by the great speed that accompanies the use of the sharp curette or adenotome. However, it does not take more than a few minutes at the most, and the surgeon can be sure of the condition of the field of operation before the patient leaves the operating room.

36 W. 40th St. (18).

REFERENCES

1. Unger, M.: Adenoid Pressure Roller for Hemostasis in Adenoidectomy, A. M. A. Arch. Otolaryng. **58**:204 (Aug.) 1953.

2. Unger, M.: Bloodless Tonsillectomy, Eye, Ear, Nose & Throat Month. **33**:38 (Jan.) 1954.

ATROPHIC RHINITIS

RICHARD L. RUGGLES, M.D.
CLEVELAND

Much has been written concerning the etiology and treatment of atrophic rhinitis. In this paper I shall present a small series of cases of this unpleasant disease on which a mode of therapy has been used that has not been described previously, insofar as I can discover.

ETIOLOGY

Lederer [1] lists many possibilities as to the cause of atrophic rhinitis, among which are that the disease follows hypertrophic rhinitis, heredity, anatomic deformities, chronic suppurative rhinitis, trauma, syphilis, and other causes which are discussed.

PATHOLOGY

Jackson and Jackson [3] state that the cause is unknown and that there is atrophy of the epithelium, glands, and vessels. Imperatori and Burman [2] suggest that there is perivascular infiltration of round cells and destruction of the nerve and organs. Lederer [1] describes a desquamation of the columnar ciliated cells and replacement by stratified squamous epithelium. There is a decrease of connective tissue, obliteration of the blood supply associated with infiltration of round cells, fibrosis, and endarteritis. There might be some relationship between this and the pathology seen in periarteritis nodosa of asthmatic patients.

SYMPTOMS

Symptoms are formation of crusting that may be foul-smelling and purulent secretions. The turbinates are small, thin, and hard. Discomfort from the crusts, changes in the associated structures of the ear and throat, and social withdrawal make the sufferer a pitiable person.

THERAPY

Treatment includes the use of local cleansing and topical applications of such substances as silver nitrate, menthol in oil, estrogenic oils, iodine preparations, ichthammol (Ichthyol), scarlet red, and many others. Surgical reduction of the nasal airway by septal implants of ivory, plastic, or bone and moving the lateral nasal wall medially by various procedures have also been advocated. Sternstein [4] recently suggested the use of such vasodilators as nicotinic acid (Niacin), histamine, and benzazoline hydrochloride (Priscol).

Since I noted that there was in some of the cases I treated an increased eosinophile count in the secretions of the nose, an attempt was made to attack the problem from an allergic background. Not all of the patients would subject themselves to testing, and not all of them were followed as long as would be desirable. Those who have used the methods of Hansel have noted that slightly greater than optimal doses of extracts of dust will temporarily increase nasal congestion; hence, it occurred to me that more than optimal doses might have the desired effect of producing turbinal edema. The mode of therapy used was a gradually increasing dosage of house dust and mold injection extract, according to Hansel's method, up to the point that the turbinates became edematous and the crusts disappeared. The dose producing the desired effect was then maintained and the time interval between injections gradually lengthened. It then became apparent that there were two groups of patients, i. e., those whose disease was reversible and thus not requiring continuous therapy and those who did well as long as the injections were

Read before the 1953 spring meeting of the Cleveland Otolaryngological Society March 18, 1953.

continued and then reverted to some extent to the original disease when therapy was stopped. The reversible group had a shorter history of the disease than did the irreversible. A brief résumé of treated cases is presented.

REPORT OF CASES

CASE 1.—F. M., a 31-year-old white man, first noticed nasal crusting in 1944, while serving with the Army in Belgium. The symptoms became worse on his return to the United States. In May, 1944, while at the Crile Veterans Hospital, he underwent an allergy work-up, which revealed multiple food, dust, and pollen sensitivities. After three months of gradually increasing doses of dust and mold extract to the point where the turbinates developed edema and lost the crusts, no further therapy was used. At the time of writing there has been no further trouble with nasal crusting.

CASE 2.—R. H., a 40-year-old white woman, had atrophic rhinitis for 20 years, with mild ozena and anosmia. She also had a small aural polyp. She was treated with local cleansing and dust and the mold injections of gradually increasing size for three weeks, at which time there was much improvement; she moved from the city at that time, and no further follow-up could be maintained.

CASE 3.—W. W., a 38-year-old Negro man, had a history of atrophic rhinitis for 10 years. Gradually increasing doses of dust and mold extract administered for three months produced marked edema of the turbinates and a decrease in the amount of crusting while the therapy was maintained. The patient when last seen was well satisfied with the result obtained.

CASE 4.—F. D., a 51-year-old white man, had had, in addition to a retinal detachment and a chronic uveitis, atrophic rhinitis for many years. In March, 1952, he had a submucous resection with insertions of an acrylic implant into the septum to alleviate his nasal difficulty. Several weeks thereafter the implant was extruded. An iliac crest graft to reconstruct the saddle nose at the same time was successfully retained, however. In June, 1952, therapy with dust and mold extract was begun, and the turbinates shortly thereafter began to swell, with a consequent disappearance of the nasal crusting. In March, 1953, he began to complain of postnasal drainage; hence the dosage was reduced. In April no further injections were necessary, and the patient has remained asymptomatic.

CASE 5.—W. M., a 60-year-old white man, had had atrophic rhinitis since World War I, with mild ozena. Three months' therapy with dust and mold extract produced turbinal edema, postnasal drainage, and gradual disappearance of the crusts.

CASE 6.—A. N., a 61-year-old white woman, had had atrophic rhinitis for 30 years, with severe crusting. After six months of therapy with dust and mold extract, her turbinates were enlarged and there was mild crusting; she seemed satisfied with the results obtained when last seen.

SUMMARY

Six cases of atrophic rhinitis are presented; the use of increasing doses of dust and mold extract to produce turbinal edema and elimination of the crusting is described. Although this is a very small series of cases, this paper is written in the hope that it may stimulate others to try this method, which is only one of many but previously undescribed so far as I can discover.

10515 Carnegie Ave.(6).

REFERENCES

1. Lederer, F. L.: Diseases of the Ear, Nose and Throat, Ed. 3, Philadelphia, F. A. Davis Company, 1942.

2. Imperatori, C. J., and Burman, H. J.: Diseases of the Nose and Throat, Ed. 3, Philadelphia, J. B. Lippincott Company, 1947.

3. Diseases of the Nose, Throat, and Ear, edited by C. Jackson and C. L. Jackson, Philadelphia, W. B. Saunders Company, 1945.

4. Sternstein, H. J.: Use of Antibiotics and Vasodilators in Atrophic Rhinitis (Ozena), New England J. Med. **245**:165 (Aug. 2) 1951.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1950, 1951, and 1952

ALLERGY OF THE EYE, EAR, NOSE, AND THROAT

ERNEST L. MacQUIDDY, M.D.
OMAHA
AND
JOHN M. SHELDON, M.D.
ANN ARBOR, MICH.

WORKERS in allergy and applied immunology have contributed a voluminous amount to the literature in the past few years. This summary includes most of the key articles which might be of interest to otorhinolaryngologists. Purposely, we have made no attempt to editorialize on certain papers which are controversial or perhaps lacking in statistical or clinical confirmation. The reader, if stimulated, should judge the article in question for himself after careful perusal of the original source. Furthermore, because of the extensive number of papers on the use of cortisone or corticotropin (ACTH) in ophthalmic conditions, this subject was reserved for a separate review.

I. GENERAL ASPECTS OF ALLERGY OF INTEREST TO OTOLARYNGOLOGISTS

Ashley,[1] in an article on modern trends in otolaryngology with special emphasis on hypersensitivity, advances the theory that allergy is a phenomenon of autonomic imbalance, resulting from various types of stimuli. Allergic symptoms referable to the nose, throat, and ear, he feels, result from increased permeability of blood vessels and tissue cells, with a corresponding imbalance of fluid and electrolytes. Williams * advances a theory of allergy based on autonomic vascular reactions, which, he feels, furnishes a better working hypothesis than one based on the antigen-antibody concept. Hansel [4] briefly enumerates the commoner areas of otolaryngologic manifestations of allergy as follows:

a. The respiratory tract—nose, paranasal sinuses, adenoids and tonsils, nasopharynx, larynx, trachea and bronchi

b. The mouth—oral mucosa, gums, lips, tongue, soft palate and salivary glands

c. The ear—auricle, canal, tympanum, middle ear, Eustachian tube, labyrinth, and cochlea

Systemic manifestations of allergy also may commonly be seen in conjunction with disturbances of one or more of the above-listed sites.† Clements [7] considers the modern trends in treatment of ear, nose, and throat allergy. He reviews the

Roy Patterson, Robert G. Lovell, and Kenneth P. Mathews contributed in the preparation of this review.

From the University of Nebraska Medical School and the Department of Internal Medicine, University of Michigan Medical School.

* References *2* and *3*.

† References 5 and 6.

methods of diagnosis and treatment, including careful history and inventory of symptoms; physical examination; microscopic study of secretions, body fluids, and soft tissues; roentgenographic examination; diagnostic tests, including skin tests; deliberate exposure to inhalants; withholding of certain foods followed by their controlled ingestion, and sometimes a therapeutic trial of hyposensitization, if it is clinically indicated. McGowan [8] stresses the importance of allergy in otolaryngology, including allergic involvement of the salivary glands and deafness due to allergy. He advises against removal of tonsils and adenoids for the relief of early respiratory allergy.

Kern [9] points out the importance of allergic disease in military medicine and observes that it often is overlooked in candidates for military service. He states that this oversight is responsible for a high rate of morbidity, invalidism, medical discharge, and ultimate compensation in the armed forces.

II. ALLERGIC CONDITIONS OF THE EAR

Increasing interest has been shown in regard to allergic manifestations in and around the ear. Dintenfass,[10] in a discussion of hypersensitivity conditions in otology, stresses early recognition and states that the incidence of allergic symptoms and pathologic variations of the ear due to altered sensitivity is much greater than generally is realized. He describes diagnosis and treatment of allergic conditions which may affect the ear and classifies them as follows:

Location	Clinical Manifestations
External ear and auditory canal	Contact dermatitis
	Eczema
	Urticaria
	Purpura
Middle ear and Eustachian tube	Conduction deafness
	Clinical otitis media
	Sensation of fullness
	Itching of the ear, back of the nose, between the nose and the ear
	Deep pain (dull or burning type)
	Sensation of tightness or drawing
Internal ear and auditory nerve	Perception deafness
	Nystagmus
	Ménière's symptom complex
	Vertigo
	Tinnitus

Jordan [11] also discusses the importance of allergy in otology. He suggests that clinical observations indicate that many body changes classified as allergic cannot be explained purely on the basis of antigen-antibody reactions. He points out that autonomic dysfunction, psychic changes, vasomotor reaction, and endocrine imbalance are equally important in the production of the observed altered reaction. A discussion of allergic manifestations of the external, middle, and inner ear is presented. In the external ear, 8 of 47 of his cases of chronic otitis externa were shown to have an allergic etiology. In the middle ear, 87% of 111 of his cases of chronic secretory otitis media were classified as allergic in origin. Determining the allergic factor, if it is present, is an important problem in otology. The diagnosis is difficult, and the complications of misdiagnosed cases, he points out, are often irreversible.

Jordan states that the diagnosis of allergy in the ear is substantiated by observing intermittent profuse serosanguineous discharge associated with allergic exacerbations. The finding of eosinophiles in this discharge has not been consistent and is not considered diagnostic. He also reports that of 61 cases of chronic suppurative otitis media with central perforations, 27 were complicated by allergic conditions. In patients with radical mastoidectomies in whom the middle ear mucosa was not removed and in whom the Eustachian tube remained open, Jordan attributes postoperative persistent discharge possibly to an allergic reaction. Postoperatively, the ears of 7 of 17 patients with persistent moist cavities became dry after allergic management. This author in addition states that allergic reactions also may complicate operation for fenestration. Allergic treatment may facilitate such surgery. He discusses polyps in the middle ear—a rare but troublesome problem—and states that they have the same etiology as nasal polyps. Jordan belongs to the group who feel that allergy may produce inner-ear disturbances, such as Ménière's disease. Some of his patients with inner-ear disease were relieved by allergic management.

Kuhn,[12] in a discussion of deafness in relation to allergy, states that allergic deafness problems are relatively easy to diagnose and can be very satisfactorily treated by allergic methods, with attention to nasal complications, either allergic or infectious. In the recognition of hearing disorders due to allergy, the best clue is observance of variability in the presence and intensity of hearing changes from day to day and from hour to hour. Diagnosis may be made from the patient's history, the variability of the symptoms over a period of time, and an audiogram. A base-line audiogram, together with a record of the patient's subjective feelings, is obtained, and this information is compared with an audiogram made 20 minutes after a hypodermic injection of one-half the usual therapeutic dose of epinephrine. If there is a subjective change noted and increase in the hearing acuity, the patient should be investigated from an allergic standpoint. Jones and his associates [13] discuss allergy and its relation to impaired hearing and sinusitis in children. They review the methods used in testing and the treatment of hearing disorders.

Cope [14] reviews allergic otitis media in an article in which he states that chronic suppurative otitis media which resists conventional therapy has occasionally responded to allergic management. He reports cases to show that allergy may be a cause for "chronic mucoid ears" and for chronic catarrhal otitis media. Success is reported after allergic treatment in drying up a number of the "mucoid ears," and the hearing has been improved in the secretory type with intact ear drums.

Some aspects of the influence of histamine on 53 cases of Ménière's disease are reported by Richtner.[15] These patients were studied with reference to histamine as an etiologic factor and as a measure for treatment of the syndrome. Histamine skin tests by this author's technique showed positive reactions in about 15% of the vertigo cases. The mean blood histamine value was lower in vertigo patients than in normal persons or in otosclerosis cases and was lowest in those presenting positive histamine skin reactions. A few patients were treated with histamine, but no definite conclusions as to the results were made.

III. ALLERGIC CONDITIONS OF THE EYE

Hansen [16] reviews allergic conditions in the field of ophthalmology and discusses the manifestations of allergy in the various ocular tissues. He regards the presence of eosinophiles in the exudates as the most significant diagnostic criterion that

allergy is the cause of such lesions involving the lids and conjunctiva. Phlyctenular conjunctivitis and vernal conjunctivitis now are considered to be allergic conditions. He also feels that many lesions of the cornea and retina can be explained on the basis of hypersensitivity reactions. The complex nature of the mechanisms of allergy of the eye and adnexa are discussed by Poos,[17] who points out that one of the main shock organs involved is the blood vessel. A fuller understanding of the allergic mechanism results in better treatment of patients, and an ophthalmologist has the advantage of being able to observe the retinal vessels during allergic reactions with an ophthalmoscope. Poos suggests that a typical allergic reaction of the antibody-antigen type is a defense reaction, anabolic in type, with parasympathetic stimuli in predominance. Thus he assumes that allergic cases will be helped through stimulation of the sympathetic system by the administration of epinephrine and by other measures which he outlines. Another group of conditions, primarily vasospastic in nature, are characterized by him as catabolic reactions and manifestations of the sympathetic nervous system stimulus. They may be helped by vasodilators. Poos reviews the concepts of allergy and immunology and also lists allergic manifestations of the eye and its adnexa.

De Muro and Focosi [18] describe their experimental studies on allergic thrombosis of the retinal vessels. Rabbits sensitized to sheep serum were shocked by vitreum inoculations. Thrombosis of retinal vessels was observed at 6- and 24-hour intervals after the exciting inoculation. These studies substantiate the possibility that allergic reactions may affect the retinal vessels.

An article by Posner [19] gives a comprehensive review of allergy of the conjunctiva. He classifies allergic conjunctivitis under two headings: (*a*) acute edematous allergic conjunctivitis, characterized by an immediate type of allergic response, and (*b*) allergic dermatoconjunctivitis, which he regards as a form of contact allergy and the more important clinical condition of the two. Theodore [20] stresses the importance of finding eosinophiles in smears of conjunctival secretions and in scrapings of the conjunctival epithelium. These cells, he feels, are almost pathognomonic of allergic conjunctivitis or vernal catarrh, if other causes of eosinophilia are ruled out. He states that a negative finding does not exclude allergic etiology. A review of the differential diagnosis of eosinophilia is presented, and a description is given of the method used for cytological examination. An epithelial scraper made from a platinum loop may be scraped, without anesthesia, over the conjunctiva to obtain material. This substance then is spread on a glass slide and stained by the Giemsa or Wright technique. The article points out that eosinophilia may be seen in the peripheral blood in parasitic diseases, periarteritis nodosa, Löffler's syndrome, mycosis fungoides, pemphigus, and allergic disease. A peripheral blood eosinophilia does not result in an eosinophilia of the conjunctiva. The author points out that certain nonallergic irritants, such as lime, ipecac, insect powder, and turpentine oil, may produce a massive conjunctival eosinophilia ranging up to 80% of all leucocytes. Lecomte [21] points out that a blood eosinophilia is not necessarily diagnostic of an allergic condition. Nonallergic causes of eosinophilia are outlined. In addition to parasitic infestation one may find an eosinophilia in the following conditions: skin diseases, especially pemphigus and dermatitis herpetiformis; during convalescence from acute infection; accompanying such diseases as periarteritis nodosa, dermato-

myositis, rheumatoid arthritis, and Löffler's syndrome; in erythema multiforme and with certain neoplasma; after irradiation; after administration of liver to patients with pernicious anemia, and as an unusual familial characteristic.

Bentolila, Ortiz, and Bertotto [22] report a case of allergic cataract and review the literature. Such lens opacities, they point out, are associated with atopic dermatitis and occur only after the dermatitis is established. In such cases both the skin and the lens apparently react as the shock organ to the exciting allergen. Senear [23] reports a case of bilateral cataracts in a boy who had atopic eczema. The patient also had asthma.

Maumenee [24] discusses the influence of donor-recipient sensitization on corneal grafts. Experiments were conducted on rabbits to obtain further information regarding the reason for the clouding of certain corneal grafts postoperatively. One cause of this clouding may be sensitization of the recipient to protein material from the donor. Definite conclusions were not drawn from the experiments, but the author feels that cortisone may be of benefit in preventing allergic complications in tissue transplantation work.

Swinny [25] describes periorbital dermatitis as the most frequent regional dermatosis in his private practice of allergy. The most frequent cause he observed to be cosmetics, with such items as cream-based shampoos, powder foundations, nail polish, and liquid dyes being particularly troublesome. Overtreatment of the dermatitis or occupational contactants also accounted for some of his cases. The author states that atopy plays little or no part in periorbital dermatitis and usually may be disregarded in the study of lesions of this area. Zakon and Goldberg [26] review the dermatoses seen on the eyelids, including those of an allergic nature. They list contact dermatitis and dermatitis medicamentosa as the two commonest lesions. Bogdanow [27] discusses cosmetics as a cause of facial dermatitis. The majority of his cases were due to hypersensitivity rather than to primary irritation. The commonest preparations causing dermatitis in his series were nail lacquers, scalp preparations, face creams, lip rouge, and perfume. For the recognition of cosmetic dermatitis, Bogdanow stresses the main diagnostic aids of the history, the clinical appearance of the lesion, and the patch test.

Christensen [28] has reported on the pathology of collagen disease in regard to the eye. He considers collagen, which is a structural tissue element, to be subject to hormonal control. Pathologic alteration of this system by a factor such as hypersensitivity reaction may give rise to an indeterminate group of systemic disturbances (collagen disease). The eye, having a high collagen content, is occasionally involved in these disturbances. Lesions observed in the eye are variable in type, site, and degree of severity, but the pathology is similar to that seen elsewhere in the body.

Further publications on the use of antihistamines in ocular diseases have appeared in the literature. Hurwitz [29] used antazoline (Antistine) phosphate ophthalmic solution in 100 patients. He reports that the drug is of decided symptomatic palliative value in 80% of those with ocular allergies or ocular itching of other origin. A great advantage of topical treatment in ocular allergy over its use in other fields of medicine is claimed by Stocker.[30] Topical administration minimized side-reactions, in his opinion, if allergic symptoms were mainly limited to the anterior segment of the eye and its adnexa. Kahan and Carter [31] describe prophenpyridamine (Trimeton) maleate as a helpful adjunct in the treatment of diseases which necessitate prolonged application of drugs capable of causing sensitivity reactions.

D'Ermo [32] and Atkinson [33] discuss the use of antihistamines in ophthalmic conditions. Atkinson points out that topical application of antihistamine to the normal eye results in pupillary dilatation, reduced capacity for accommodation, and possible blurred vision. Stoesser [34] has reported a study of the use of antihistamines in over four thousand allergic children. He suggests a division of antihistaminic drugs into five groups, based on their chemical characteristics and the side-reactions produced. He suggests that doxylamine (Decapryn) succinate, diphenhydramine and aminophylline (Hydryllin), and diphenhydramine (Benadryl) hydrochloride—ethanolamines—be regarded as likely sedatives, while phenindamine tartrate (Thephorin) may stimulate. The other antihistamines fall in between the two extremes, but individual idiosyncrasy must be kept in mind. A schedule of clinical activity for the drugs is presented, and the use of the compounds in various allergic manifestations is described. Sherman [35] likewise presents a classification of the histamine antagonists. Dannenberg and Feinberg [36] report on the development of tolerance to the antihistamines. These authors believe that clinically such tolerance is only relative and that most patients will continue to obtain relief from prolonged therapy if dosage is increased or if the drug is discontinued for from 3 to 14 days.

A case of agranulocytosis after prolonged use of tripelennamine (Pyribenzamine) hydrochloride is reported by Martland and Guck,[37] and a similar report appears by Hilker.[38] Yapalater and Rockwell [39] present a case of toxic psychosis, which they attribute to prophenpyridamine, with electroencephalograms showing abnormal readings after administration of chlorprophenpyridamine (Chlor-Trimeton). Crumbley [40] reports three instances of hemolytic anemia apparently resulting from the administration of diphenhydramine and tripelennamine.

Ferris and his associates [41] suggest that allergic transfusion reactions may be prevented by the prophylactic use of 25 mg. of tripelennamine in each pint of blood transfused. In 607 transfusions there was one allergic reaction and no pyrogenic effects. In the control series of 742 pints of blood given without the antihistamine, there were 20 allergic reactions and 32 pyrogenic reactions.

IV. NASAL ALLERGY

The preventive aspects of pediatric allergy are emphasized by Frazier.[42] He considers early recognition and treatment of allergic disease in children an important way to avoid complications. Glaser, Kaiser, and Siegel [43] discuss ragweed pollinosis as a public health problem and in a survey of 2,140 school children found an incidence of hay fever of 8.3%. A larger detailed survey is recommended. Allergy as a factor in childhood sinobronchitis is discussed by Price and Solow.[44] Tabulations of the prevalence and degree of sinobronchitis were correlated with allergic factors in a selected patient group. The authors found that in allergic children 5 years of age or under who were hospitalized for chronic respiratory disease, there was demonstrated roentgenographic evidence of sinobronchial disease in 64%. This group was studied for susceptibility to lower respiratory tract infection. Grove [45] stresses the importance of hyperplastic sinusitis, either primary or secondary, as an etiologic factor in such diseases as vasomotor rhinitis, urticaria, angioneurotic edema, eczema, and bronchial asthma. He discusses the part which bacterial allergy may play in certain of these conditions. Shambaugh [46] discusses allergy in relation to chronic sinusitis. He states that no more than one-fourth of all cases of chronic suppurative sinusitis are of the anaerobic nonallergic type. The great majority, he

feels, have an allergic basis in which chronic allergy of the nasal sinus mucosa is complicated by a superimposed infection. These cases, he points out, do not respond to nasal and sinus surgery unless the underlying allergy is suspected and controlled. Jones [47] reviews the case records of 3,143 otolaryngological patients, 522 of which showed positive evidence of allergy. He discusses a program of into-allergic measures and the necessary nasal surgery in carefully selected cases.

The part which food sensitivity plays in nasal allergy has been reviewed by Davison,[48] who discusses its etiological importance in the production of allergic reactions. The many nonpollen inhalant substances which frequently cause nasal allergy are discussed by Prince.[49] This paper includes a listing and classification of environmental and occupational inhalants, including dusts, danders and hair, insecticides, cosmetics, seeds, and molds. The management of mold allergy by avoiding heavy concentrations and likely sources of spores is described.

In an article on hay fever, humidity, and sinusitis, M. Unger [50] advances his belief that hay fever is not an entirely separate disease caused by pollen sensitivity but is an acute exacerbation of chronic nasosinusitis, which is induced by the excessive humidity of the season.

The relationship between the presence of tonsils and the existence of nasal allergy is described by Goodman,[51] who states that hypertrophy of lymphoid tissue in the nasopharynx is much commoner in allergic than in nonallergic children. Removal of this tissue in patients under 5 years of age or removal without the institution of allergic management usually is followed by prompt recurrence of lymphoid tissue.

Millonig, Harris, and Gardner [52] describe the effect of autonomic denervation of nasal mucosa. Surgical interruption of the sympathetic nervous system supplying the mucous membranes of the nose was followed by nasal stuffiness and discharge in 27 cases. These changes, the authors suggest, were the result of a loss of vasoconstrictor tone. In six patients in whom the parasympathetic nerve supply to the nasal mucous membrane was interrupted, the exact opposite effects were noted: the mucous membranes were dry and shrunken. The similarity between the appearance of the nasal mucosa following sympathectomy and that observed in allergic rhinitis suggests that there may be a close association between the autonomic system and allergic disease.

Fox and Niles [53] present a new classification of chronic rhinosinusitis which combines the pathologic condition with a clinical term describing the dominant symptom. Melchior,[54] in an extensive report, discusses allergy and vasomotor disturbances of the nasal and sinusal mucosa. The first five chapters of this report deal with the history of allergy, experimental anaphylaxis, infectious allergy, nonspecific allergy, and the traits peculiar to allergy in humans. The last three chapters describe vasomotor disturbances of nasosinusal mucosa, polyposis, noninfectious nasal allergy, and infectious nasosinusal allergy.

Stevens and Gordon [55] review the etiology and treatment of bacterial respiratory infections resembling epidemic coryza in allergic patients. In their opinion potentially allergic children have numerous respiratory infections apparently not of virus etiology but which are recurrences of bacterial infection. Continued inoculations with filtrates of cultures of upper respiratory tract bacteria appear to control these infections. Blatt and Nantz [56] identify the offending organism in bacterial allergy by means of a series of leucolytic tests, challenging the patient's leucocytes with various bacterial filtrates. Patients then are treated by the administration of bacterial filtrates

to which they have shown positive reactions. The authors advise careful dosage, as systemic reactions are easily provoked. Many of their cases in which routine treatment was unsuccessful experienced excellent results when treated with bacterial filtrates. Bacterial infection in respiratory allergy also is discussed by Silcox.[57] He studied 110 patients with respiratory allergy who had concomitant infection. Ninety of these persons treated with bacterial vaccine for one year reported improvement. The importance of nonallergic factors in hypersensitivity disease is stressed by Hollender, Fabricant, and Posner.[58] Such nonallergenic environmental factors as climate, season, and weather may influence the clinical symptoms. Social status, habits, and the presence of infection in addition to endocrine influence are mentioned. Physical, chemical, and psychic agents also affect the picture, and it is urged that the nonallergic items be considered in addition to the allergens when one is studying a case of sensitization.

Wittich[59] reports the effect of air pollution on respiratory tract allergy. He states that rusts, smuts, and organic material disseminated from manufacturing plants as well as chemical substances may act as specific allergens to produce allergic diseases. Symptoms of hay fever also may be caused by the algae found in lakes in certain areas during August and September, according to Heise.[60]

Harris and Shure[61] report a case of fatal allergic shock following intradermal tests for allergy, with a description of the pathological anatomy. This consisted primarily of acute pulmonary emphysema of the so-called "guinea pig type" of anaphylactic reaction. Cases of anaphylactic deaths in the literature are reviewed, and the advisability of preceding intradermal tests with scratch tests is emphasized. Biederman[62] describes a method for making inhalant allergy tests which enables the physician to test for volatile as well as particulate allergens. A description of the instrument and its advantages is presented. A mechanical scarifier for precision scratching in allergy testing is described by Morrison.[63] This is a cylinder with two steel points which can be pressed and rotated against the skin to produce a circular scratch. Hajos[64] reports positive skin reactions in several of 91 allergic patients tested with progesterone, testosterone, and desoxycorticosterone.

An extensive discussion of the theory and method for treating patients with nasal allergy is presented by Slepian.[65] Thacker[66] reviews the various nonspecific methods of treating nasal allergy and discusses a number of therapeutic agents. These include topical sympathomimetic vasoconstrictors such as phenylephrine (Neo-Synephrine) hydrochloride, ephedrine hydrochloride, and naphazoline (Privine) hydrochloride. He advises the use of inhalers, such as propylhexedrine (Benzedrex), only every one or two hours because of the hazard of overdose. Oral drugs, including antihistamines, ephedrine, barbiturates, ascorbic acid, and corticotropin and cortisone are mentioned as useful in selected cases. Naterman[67] describes a method for treating hay fever by the use of pollen tannate suspended in peanut oil with aluminum monostearate. His results after treating 122 patients with pollen tannate in this manner were 90% satisfactory. Management of seasonal hay fever is reviewed by Taub,[68] who describes the method and importance of correct diagnosis, the symptomatic treatment of seasonal hay fever, and the specific measures to be employed. The procedures of preseasonal, coseasonal, and perennial hyposensitization are described along with a discussion of the causes of failure in treatment of seasonal pollinosis, including inadequate or incomplete testing; overlooking con-

comitant dust, mold, or food allergy; using extract which has lost its potency; employing commercial pollen extracts not prepared specifically according to the clinical needs of the patient concerned; failing to start pollen injections early enough, and discontinue treatment after the first season rather than proceeding through several seasons. Fabricant [69] describes the drugs, including corticotropin and cortisone, used for systemic medication in nasal allergy. Benefit from iontophoresis of tripelennamine was obtained in eight patients with allergic rhinitis who had not received relief from oral antihistamine therapy by Aaron.[70] Slepian [71] reviews the management of hay fever by hyposensitization. The causes, seasons, and methods of testing and treatment are presented. Black and Holman [72] advocate oral treatment with dust and pollen extracts of respiratory allergy in children. The method is described as simple and was found to be successful in more than one-half of the 402 children using it. Hypodermic therapy gave satisfactory relief in a large number of those who failed to benefit from the oral method. Wittich [73] reports the use of a pure carbohydrate derivative from certain bacteria (Pyromen) in the treatment of perennial respiratory allergy. He finds this material to have been helpful in some of the 50 cases he treated. Samter and Kofoed,[74] on the other hand, report that Pyromen fails to produce a demonstrable decrease in formation of antibodies or depression of antigen-antibody reactions in guinea pigs. They felt in 38 allergic patients that this material was no more effective than a placebo containing its solvent.

Mathov [75] conducted an experiment on allergy to cold in the respiratory system. Forty patients with allergic symptomatology were exposed to cold water by immersion of the hands and forearms. Subjective responses and changes in the eosinophile counts on nasal secretions were observed. Forty per cent of these persons when exposed to cold suffered respiratory symptoms, and 20% were found to have a significant rise in nasal eosinophile counts. The author concludes that respiratory symptoms in those cases resulting from cold exposure are due to a truly allergic mechanism and not to a vasomotor reflex. Allergy to cold as an occupational disease also was investigated by Mathov,[76] who observed 100 workmen in a meat-packing factory. A high percentage of those men who had symptoms on exposure to cold also had eosinophilia.

Rhinitis medicamentosa is reviewed by Walker.[77] This condition accounts for nasal obstruction much more often than is suspected and results from the overuse of the nasal application of vasoconstrictors. Diagnosis is made by exclusion of other causative factors and by obtaining a history of prolonged nasal medication in addition to observing constant nasal obstruction and poor shrinkage of nasal mucous membranes on application of the usual vasoconstrictors. Stromme [78] describes a method for determining eosinophile counts of antral sinus washings. This procedure is recommended as a useful diagnostic aid in selected cases of allergic antral sinusitis. The "solid" portion of an antral washing is collected in a tube and dissolved in 8.7% saccharose in isotonic sodium chloride solution. This mixture is slowly turned for a few minutes and then centrifuged at 2,500 rpm. The lower sediment then is transferred to a pipette tube. To this is added 1 cc. of serum and 2 drops of Duboin's fixation solution. (This is made up of 20 cc. saturated sublimate solution, 5 cc. glacial acetic acid, and 1 cc. formalin.) The suspension is carefully shaken, then centrifuged 20 to 30 minutes at 3,500 rpm. The bottom sediment plug then is drained off at the lower end of the tube, fixed in 95% alcohol, and embedded in

paraffin. Five-μ-thick sections are cut, stained in May-Grünwald and Giemsa stain, and examined microscopically. The cells are easily seen and are stained so as to make identification easy.

The surgical management of nasal and sinus allergy is reviewed by van Alyea.[79] He states that successful management of allergic rhinosinusitis and polyposis is contingent upon close cooperation between the allergist and rhinologist. Seltzer,[80] in an article on plastic surgery of the nose in the allergic patient, discusses indications for surgery and irradiation and concludes that surgery alone does not cure nasal allergic reactions. He advocates hyposensitization as the chief approach but states that where obstruction of the intranasal airway is caused by bony, cartilaginous, or mucous membrane growth, such mechanical obstruction is best removed to assure better aeration. The surgery should be performed at a time other than the season during which allergic symptoms occur. Loewy and Sokolsky [81] discuss the management of choanal polyps, which are believed to have the same etiology as other nasal polyps. Nasal obstructive symptoms are variable and frequently not in proportion to the findings. Enlargement of the tissue mass may result in other symptoms. Diagnosis depends on recognizing the true nature of the growth and in differentiating the polyp from other tumors. The authors feel that surgical removal is the treatment of choice.

Reports on the use of corticotropin and cortisone in the treatment of nasal allergy are prominent in the literature. Oral cortisone therapy combined with hyposensitization in ragweed hay fever produces greater relief than that obtained previously with any other treatment. Most authors feel that the results justify the use of cortisone in selected cases after the usual therapeutic measures have failed. Carryer and his associates,[82] the Friedlaenders,[83] and Gelfand ‡ report marked symptomatic relief in patients with severe ragweed allergy treated by oral cortisone. Feinberg and his co-workers,[84] Cooke and his associates,[85] and Zeller [86] treated ragweed sensitive patients with corticotropin and cortisone. Varying but gratifying results were obtained by this treatment. Dill and Bolstad [87] used cortisone in the form of a nasal spray in the treatment of 25 patients with allergic rhinitis. Forty-eight per cent showed marked relief of symptoms, and no undesirable side-effects were observed. Using cortisone and corticotropin in the management of nasal allergy, Koelsche and his associates [88] found that in patients failing to respond to the usual methods, appreciable symptomatic relief without a severe side-effect was obtained in most cases.

V. MISCELLANEOUS PRINCIPLES

Some common misconceptions of allergic management are described by Feinberg,[89] who believes that much of the failure in allergic treatment of patients is due to misapplication of known principles. Such faulty concepts are (*a*) that every positive skin test indicates clinical allergy or merits starting desensitization; (*b*) that negative skin tests indicate an absence of allergy; (*c*) that desensitization treatment sets can be made up by a laboratory without knowledge of the clinical facts about a patient; (*d*) that desensitization is a short term "cure"; (*e*) that nonallergic factors can be ignored in allergic disease; (*f*) that the newest therapy, such as anti-

‡ Gelfand, M. L.: Use of Small Maintenance Doses of Cortisone in the Treatment of Bronchial Asthma and Hay Fever, Program of the Seventh Annual Meeting of the American Academy of Allergy, New York, Feb. 7, 1951.

histamine or hormone therapy, always is the best. These mistaken ideas can be corrected only by education, and suggestions toward this end are presented by the author.

Hypersensitivity after contact with streptomycin is discussed by Cohen and Glinsky [90] and Mitchell.[91] This is a rather common occurrence among those administering or receiving the drug. The manifestations may be drug fever, rash, urticaria, or exfoliative dermatitis. Before a patient is started on streptomycin, these workers suggest that a history of previous exposure be obtained and, if positive, further sensitivity tests be carried out. Kutscher and his co-workers [92] report a controlled study of reactions following the use of oxytetracycline (Terramycin) troches. An incidence of 53.2% was noted in 143 patients. The authors warn that this drug should not be used promiscuously. Anaphylactic reaction from the local use of penicillin instilled into the maxillary sinuses is reported by Everett.[93] Antiallergic measures provided relief.

Lane and Luikart [94] review 107 cases of dermatitis from sensitivity to local anesthetics. Such epidermal hypersensitivity occurs frequently among the general population as well as in dentists and physicians. The types of lesions are described in addition to agents that most commonly provoke the reactions. Included as common sensitizers are, in their order of importance, procaine (Novocaine), orthoform, butamben (Butesin) picrate, tetracaine (Pontocaine), dibucaine (Nupercaine), ethyl aminobenzoate (Benzocaine), butacaine sulfate (Butyn), a procaine-type compound (Apothesine), 3-diethylamino-2,2-dimethylaminobenzoate (Larocaine), and piperocaine. The authors warn that these drugs should be prescribed and used with caution, since they produce a high incidence of sensitization.

From the previous articles it can be seen that progress in the field of allergy and applied immunology has been steady and gratifying.

REFERENCES

1. Ashley, R. E.: Modern Trends in Otolaryngology with Special Emphasis on Allergy, A. M. A. Arch. Otolaryng. **55**:253, 1952.

2. Williams, H. L.: A Concept of Allergy as Autonomic Dysfunction Suggested as an Improved Working Hypothesis, Tr. Am. Acad. Ophth., p. 123, 1950.

3. Williams, H. L.: A Concept of Allergy as Autonomic Dysfunction Suggested as an Improved Working Hypothesis, Ann. Otol. Rhin. & Laryng. **60**:122, 1951.

4. Hansel, F. K.: Allergy in Otolaryngology: Historical Review, Tr. Am. Acad. Ophth., Supp., p. 3, 1951.

5. Randolph, T. G.: Allergic Factors in the Etiology of Certain Mental Symptoms, J. Lab. & Clin. Med. **36**:977, 1950.

6. Rinkel, H. J.; Randolph, T. G., and Zeller, M.: Food Allergy, Springfield, Ill., Charles C Thomas, Publisher, 1951.

7. Clements, C.: Trends and Treatment in Ear, Nose, and Throat Allergy, J. M. Soc. New Jersey **48**:470, 1951.

8. McGowan, J. F.: Allergic Manifestations in Otolaryngology, North Carolina M. J. **13**:137, 1952.

9. Kern, R. A.: The Allergic Diseases in Military Medicine, Mil. Surgeon **106**:95, 1950.

10. Dintenfass, A.: Allergic Manifestations in Otology, Eye, Ear, Nose & Throat Month. **30**:647, 1951.

11. Jordan, R. E.: Deafness Due to Allergy, Laryngoscope **60**:152, 1950.

12. Kuhn, H. A.: Allergy in Otology, Ann. Allergy **9**:213, 1951.

13. Jones, M. F.; Sisson, G. A.; Bellucci, R. J., and Edmonds, F. C., Jr.: Anti-Allergic Methods Used in the Restoration of Hearing During Childhood, Ann. Otol. Rhin. & Laryng. **60**:1085, 1951.

14. Cope, B. A.: Allergic Otitis Media, Eye, Ear, Nose & Throat Month. **29**:364, 1950.

15. Richtner, N. G.: Some Aspects of Histamine in Ménière's Disease, A. M. A. Arch. Otolaryng. **55**:434, 1952.

16. Hansen, E. W.: Mucocutaneous Eruptions, Including Conjunctiva, Due to Drug Sensitivity: Etiological Similarity to Pemphigus, Tr. Am. Acad. Ophth., p. 395, 1951.

17. Poos, E. E.: Mechanism of Allergy of the Eye and Adnexa, Eye, Ear, Nose & Throat Month. **30**:471, 1951.

18. De Muro, P., and Focosi, M.: Vascular Allergy: Experimental Studies on Allergic Thrombosis of the Retinal Veins, J. Allergy **22**:114, 1951.

19. Posner, A.: Allergy of the Conjunctiva, Eye, Ear, Nose & Throat Month. **30**:678, 1951.

20. Theodore, F. H.: The Significance of Conjunctival Eosinophilia in the Diagnosis of Allergic Conjunctivitis, Eye, Ear, Nose & Throat Month. **30**:653, 1951.

21. Lecomte, J.: Eosinophilia and Diagnosis of Allergic Disorders, Rev. méd. liège **6**:682, 1951.

22. Bentolila, L.; Ortiz, V., and Bertotto, E. V.: Allergic Cataract, Ann. Allergy **10**:36, 1952.

23. Senear, F. E.: Atopic Dermatitis with Bilateral Cataracts, A. M. A. Arch. Dermat. & Syph. **63**:162, 1951.

24. Maumenee, A. E.: The Influence of Donor-Recipient Sensitization on Corneal Grafts, Am. J. Ophth. **34**:142, 1951.

25. Swinny, B.: Periorbital Dermatitis, Ann. Allergy **9**:774, 1951.

26. Zakon, S. J., and Goldberg, A. L.: The Dermatoses of the Eyelids, Eye, Ear, Nose & Throat Month. **30**:312, 1951.

27. Bogdanow, D. N.: Dermatitis from Cosmetics, Eye, Ear, Nose & Throat Month. **31**:306, 1952.

28. Christensen, L.: The Pathology of Collagen Disease Applied to the Eye, Tr. Am. Acad. Ophth., p. 536, 1951.

29. Hurwitz, P.: Local Antihistaminic Agents in Ophthalmology, Illinois M. J. **98**:113, 1950.

30. Stocker, F. W.: The Use of Antihistamine Drugs in Ophthalmology, South. M. J. **43**:242, 1950.

31. Kahan, E., and Carter, G. Z.: Trimeton Maleate in Allergic Eye Manifestations, J. M. Soc. New Jersey **48**:510, 1951.

32. D'Ermo, F.: Antihistamine Drugs and the Eye: VI. Synthetic Antihistamines in Allergic Eye Disease, Boll. ocul. **30**:135, 1951.

33. Atkinson, G. S.: Antihistamines—Their Role in Ophthalmology, M. Times **79**:415, 1951.

34. Stoesser, A. V.: Allergy in Children and the Antihistamines, Journal-Lancet **71**:83, 1951.

35. Sherman, W. B.: The Uses and Abuses of Antihistamine Drugs, Bull. New York Acad. Med. **27**:309, 1951.

36. Dannenberg, T. B., and Feinberg, S. M.: The Development of Tolerance to Antihistamines, J. Allergy **22**:330, 1951.

37. Martland, H. S., Jr., and Guck, J. K.: Agranulocytosis After Antihistamine Therapy: Report of a Case Following the Prolonged Use of Tripelennamine (Pyribenzamine Hydrochloride), J. A. M. A. **143**:742, 1950.

38. Hilker, A. W.: Agranulocytosis from Tripelennamine (Pyribenzamine Hydrochloride), J. A. M. A. **143**:741, 1950.

39. Yapalater, A. R., and Rockwell, F. V.: Toxic Psychosis Due to Prophenpyridamine (Trimeton): Report of a Case, J. A. M. A. **143**:428, 1950.

40. Crumbley, J. J., Jr.: Anemia Following Use of Antihistaminic Drugs, J. A. M. A. **143**:726, 1950.

41. Ferris, H. E.; Alpert, S., and Coakley, C. S.: Prevention of Allergic Transfusion Reactions: The Prophylactic Use of Antihistamine in Blood to Prevent Allergic Transfusion Reactions, Am. Pract. & Digest Treat. **3**:177, 1952.

42. Frazier, C. A.: Preventive Aspects of Pediatric Allergy, North Carolina M. J. **12**:180, 1951.

43. Glaser, F.; Kaiser, A. D., and Siegel, S. C.: Ragweed Pollinosis (Hay Fever)—A Public Health Problem in School Children, Ann. Allergy **9**:226, 1951.

44. Price, W. C., and Solow, A. I.: Allergy as a Factor in Childhood Sinobronchitis, J. Allergy **22**:4, 1951.

45. Grove, R. C.: The Importance of Hyperplastic Sinusitis in Bacterial Allergy, J. Allergy **22**:550, 1951.

46. Shambaugh, G. E.: Allergy in Relation to Chronic Sinusitis, Graduate Lecture Course 491, Tr. Am. Acad. Ophth., p. 49, 1950.

47. Jones, M. F.: Allergy of the Upper Respiratory Tract: Cure by Surgery Plus Antiallergic Methods, A. M. A. Arch. Otolaryng. **55**:261, 1952.

48. Davison, H. M.: The Role of Food Sensitivity in Nasal Allergy, Ann. Allergy **9**:568, 1951.

49. Prince, H. E.: Miscellaneous Inhalants and Molds, Ann. Allergy **9**:575, 1951.

50. Unger, M.: Hay Fever, Humidity and Sinusitis, New York J. Med. **50**:1727, 1950.

51. Goodman, E. G.: Present Day Treatment of Allergic Conditions, North Carolina M. J. **12**:5, 1951.

52. Millonig, A. F.; Harris, H. E., and Gardner, W. J.: Effect of Autonomic Denervation on Nasal Mucosa, Arch. Otolaryng. **52**:359, 1950.

53. Fox, N., and Niles, L. M.: Classification of Chronic Diseases of Nose and Accessory Sinuses, Arch. Otolaryng. **52**:900, 1950.

54. Melchior, R.: Allergy and Vasomotor Disturbances of the Nasal and Sinusal Mucosa, Acta oto-rhino-laryng. belg. **4**:5, 1950.

55. Stevens, F. A., and Gordon, E.: Occurrence of Bacterial Respiratory Infections Resembling Epidemic Coryza in Allergic Patients: Etiology and Treatment, Ann. Allergy **9**:634, 1951.

56. Blatt, H., and Nantz, F. A.: Sinusitis Due to Bacterial Allergy: Identified by Tissue Culture and Treated by Specific Desensitization, Ohio M. J. **46**:319, 1950.

57. Silcox, L. E.: The Role of Bacterial Infection in Respiratory Allergy, Laryngoscope **60**:225, 1950.

58. Hollender, A. R.; Fabricant, N. D., and Posner, A.: Editorial: Non-Allergic Factors in Allergic Disease, Eye, Ear, Nose & Throat Month. **30**:316, 1951.

59. Wittich, F. W.: Respiratory Tract Allergic Effects from Chemical Air Pollution, Arch. Indust. Hyg. **2**:329, 1950.

60. Heise, H. A.: Symptoms of Hay Fever Caused by Algae: II. Microcystis, Another Form of Algae Producing Allergenic Reactions, Ann. Allergy **9**:100, 1951.

61. Harris, M. C., and Shure, N.: Sudden Death Due to Allergy Tests, J. Allergy **21**:208, 1950.

62. Biederman, J. B.: A New Method for Making Allergy Tests, Eye, Ear, Nose & Throat Month. **29**:558, 1950.

63. Morrison, I. R.: Precision Scratching with a Micro-Scratcher, J. Allergy **23**:187, 1952.

64. Hajos, M. K.: Skin Tests with Steroid Hormones in Allergic Diseases, Ann. Allergy **8**:66, 1950.

65. Slepian, S.: Specific Immunization for Nasal Allergy, Eye, Ear, Nose & Throat Month. **30**:656, 1951.

66. Thacker, E. A.: Non-Specific Therapeutic Methods for Nasal Allergy, Eye, Ear, Nose & Throat Month. **30**:662, 1951.

67. Naterman, H. L.: Pollen Tannate Suspended in Peanut Oil with Aluminum Monostearate in the Treatment of Hay Fever, J. Allergy **22**:175, 1951.

68. Taub, S. J.: Management of Seasonal Hay Fever, Eye, Ear, Nose & Throat Month. **30**:668, 1951.

69. Fabricant, N. D.: Systemic Medication in Nasal Allergy, Eye, Ear, Nose & Throat Month. **30**:676, 1951.

70. Aaron, T. H.: Iontophoresis of Pyribenzamine in Allergy Rhinitis, Canad. M. A. J. **61**:301, 1949.

71. Slepian, S.: Treatment of Hay Fever by Hyposensitization, A. M. A. Arch. Otolaryng. **54**:157, 1951.

72. Black, J. H., and Holman, J.: Treatment of Respiratory Allergy in Children by Oral Administration of Dust and Pollen Extracts, J. Allergy **21**:148, 1950.

73. Wittich, F. W.: Pyromen in the Treatment of Perennial Respiratory Allergies, Ann. Allergy **9**:502, 1951.

74. Samter, M., and Kofoed, M. A.: On the Rationale of Treating Allergic Diseases with Bacterial Pyrogens, J. Allergy **23**:327, 1952.

75. Mathov, E.: Allergy to Cold in the Respiratory System: Characteristics and Incidence in the Allergic Patient; An Experimental Study, Ann. Allergy **8**:366, 1950.

76. Mathov, E.: Allergy to Cold as an Occupational Disease: Clinical and Experimental Study on 100 Workmen in Meat-Packing Factory, Ann. Allergy **8**:373, 1950.

77. Walker, J. S.: Rhinitis Medicamentosa, J. Allergy **23**:183, 1952.

78. Stromme, O.: A Cyto-Histological Method as a Diagnostic Aid in Allergic Antral Sinusitis, Ann. Allergy **8**:362, 1950.

79. van Alyea, O. E.: Surgical Management of Nasal and Sinus Allergy, Ann. Allergy **9**:585, 1951.

80. Seltzer, A. P.: Plastic Surgery of the Nose in the Allergic Patient, Eye, Ear, Nose & Throat Month. **29**:687, 1950.

81. Loewy, A., and Sokolsky, I.: Management of Choanal Polyps, Eye, Ear, Nose & Throat Month. **31**:199, 1952.

82. Carryer, H. M., and others: The Effect of Cortisone on Bronchial Asthma and Hay Fever Occurring in Subjects Sensitive to Ragweed Pollen, J. Allergy **21**:282, 1950.

83. Friedlaender, S., and Friedlaender, A. S.: Oral Cortisone Therapy in Allergic Diseases, J. Allergy **22**:291, 1951.

84. Feinberg, S. M.; Dannenberg, T. B., and Malkiel, S.: ACTH and Cortisone in Allergic Manifestations, J. Allergy **22**:195, 1951.

85. Cooke, R. A., and others: ACTH and Cortisone in Allergic Diseases, J. Allergy **22**:211, 1951.

86. Zeller, M.: ACTH Effect on Ragweed Hay Fever, Ann. Allergy **9**:603, 1951.

87. Dill, J. L., and Bolstad, D. S.: Observations on the Local Use of Cortisone in the Nose in Allergic Rhinitis, Laryngoscope **61**:415, 1951.

88. Koelsche, G. A.; Maytum, C. K.; Prickman, L. E., and Carryer, H. M.: Use of Cortisone and ACTH in the Management of Nasal Allergy, Ann. Allergy **9**:573, 1951.

89. Feinberg, S. M.: Allergy Therapy: Some Common Misconceptions of Allergic Management, J. A. M. A. **147**:617, 1951.

90. Cohen, A. C., and Glinsky, G. C.: Hypersensitivity to Streptomycin, J. Allergy **22**:63, 1951.

91. Mitchell, H. S.: Streptomycin Dermatitis, J. Allergy **22**:71, 1951.

92. Kutscher, A. H.; Budowsky, J.; Lane, S. L., and Chilton, N. W.: Reactions Following the Use of Terramycin Troches: A Controlled Study, J. Allergy **23**:177, 1952.

93. Everett, R.: Anaphylactic Reactions from Local Use of Penicillin: Report of 2 Cases, J. A. M. A. **146**:1314, 1951.

94. Lane, C. G., and Luikart, R. H., II: Dermatitis from Local Anesthesia with Review of 107 Cases from Literature, J. A. M. A. **146**:717, 1951.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF MAY 1, 1954

Acousticon Models A-17, A-180, & A-185
Mfr., Dictograph Products Inc.
95-25 149th St.
Jamaica 35, N. Y.

Audiotone Models 11 & 15
Mfr., Audio Company of America
5305 N. 6th St.
Phoenix, Ariz.

Audivox Models Super 67 & 70
Mfr., Audivox, Inc.
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"
Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Cleartone Models 500 & 700

Cleartone Regency Model
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Dahlberg Model D-3 (Tru-Sonic)

Dahlberg Model D-4 (Tru-Sonic)
Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Fortiphone Models 19LR, 20A, 21C, & 22
Mfr., Fortiphone Limited
Fortiphone House
247 Regent St.
London, W.1, England

Distr., Anton Heilman
75 Madison Ave.
New York 17

Gem Model V-60
Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corp.
708 W. 40th St.
Minneapolis 8

Maico Maxitone

Maico Model J

Maico Top Secret Model L
Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Micronic Model 303

Micronic Model Mercury

Micronic Star Model
Mfr., Audivox, Inc—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Classic Model T-9

Microtone Models T-10 & T-612
Mfr., The Microtone Corporation
Ford Parkway on the Mississippi
St. Paul 1

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C

Normatone Model D-53

Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Models B-15 & B-30

Otarion Models F-1 & F-3

Otarion Model G-3

Otarion Model H-1, Custom "5"
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)

Paravox Model Y (YM, YC, & YC-7)
(Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Model 62 Starlet

Radioear Model 72

Radioear Model 82 (Zephyr)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16

Distr., Radioear Corporation
306 Beverly Rd., Mt. Lebanon
Pittsburgh 16

Silvertone Models H-16, J-92, & P-15
Mfr., Johnston Hearing Aid Mfg.
Company
708 W. 40th St.
Minneapolis 8

Distr., Sears, Roebuck & Co.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940, 966, 977, & 988
Mfr., Sonotone Corporation
Elmsford, N. Y.

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 99, 200, 300-B, 400, 500, 952, 953, & 1700
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model
Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Zenith "Regent"
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All aforementioned hearing aids have vacuum tubes.)
Accepted hearing aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED

Acousticon Model A-300	1 transistor and 2 tubes
Acousticon Model A-310	1 transistor and 2 tubes
Acousticon Model A-330 Mfr., Dictograph Products Inc. 95-25 149th St. Jamaica 35, N. Y.	3 transistors
Audivox Model 71 Mfr., Audivox, Inc. 123 Worcester St. Boston 18	3 transistors
Beltone Concerto Model Mfr., Beltone Hearing Aid Co. 2900 W. 36th St. Chicago 32	3 transistors
MaicoTransist-Ear Model O Mfr., The Maico Company, Inc. 21 N. 3rd St. Minneapolis 1	3 transistors
Micronic "All American" Hearing Aid Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division 123 Worcester St. Boston 18	3 transistors
Microtone Model T1 (Red Dot)	3 transistors
Microtone Model T1 (Yellow Dot)	3 transistors
Microtone Model T31 (Micro-Mite) Mfr., The Microtone Corporation Ford Parkway on the Mississippi St. Paul 1	1 transistor and 2 tubes
Otarion Model C-15	1 transistor and 2 tubes
Otarion Model F-22 Mfr., Otarion, Inc. 4757 N. Ravenswood Ave. Chicago 40	1 transistor and 2 tubes
Radioear Model 820 Mfr., E. A. Myers & Sons, Inc. 306 Beverly Rd. Mt. Lebanon Pittsburgh 16	3 transistors
Silvertone Model H-25 Mfr., The Dahlberg Co. Golden Valley Minneapolis 22 Distr., Sears, Roebuck & Co. 925 S. Homan Ave. Chicago 7	3 transistors

Sonotone Model 1010
Mfr., Sonotone Corporation
Elmsford, N. Y.
1 transistor and 2 tubes

Telex Model 954
Mfr., Telex, Inc.
Telex Park
St. Paul 1
1 transistor and 2 tubes

Unex Model TR-3D
Mfr., Nichols & Clark
Hathorne, Mass.
3 transistors

Zenith Model Royal-T
3 transistors

Zenith Model Super Royal-T
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39
3 transistors

Semiportable Hearing Aids Accepted

Ambco Hearing Amplifier (Table Model)
Mfr., A. M. Brooks Co.
1222 W. Washington Blvd.
Los Angeles 7

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Model Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

Treatment of Otitis Externa and Chronic Media with Sodium Sulfacetamide (Sodium Sulamyd) Solution, 30%. J. D. Maddox, Eye, Ear, Nose & Throat Month. **32**:571 (Oct.) 1953.

Maddox reports the use of sodium sulfacetamide (Sodium Sulamyd) solution, 30%, and Sulzberger powder in the treatment of otitis externa and chronic otitis media. Of 29 cases with otitis media, 20 cleared completely, 2 had occasional recurrences, which responded to the same treatment, 3 were improved, 2 responded to treatment but had relapses due to acute allergy, and 2 showed no improvement. Of 76 patients with otitis externa, 42 showed excellent improvement in one week; 20 required oral sulfonamide therapy for three days and local therapy for three weeks; 8 required concomitant allergic treatment; 8 cases had recurrences, which responded to the same therapy, and 4 cases were improved only. Only two cases showed local allergic reactions to the sodium sulfacetamide solution, 30%, an incidence of only 1.9% in this series of 105 cases treated over a period of five years.

Jennes, Waterbury, Conn.

Evaluation of Antibiotic Therapy of Acute Otitis Media. M. H. Rutherford, Eye, Ear, Nose & Throat Month. **32**:579 (Oct.) 1953.

The rational therapy of acute otitis media is based on sound surgical and medical principles. The empirical use of antibiotic medication is not a substitute for such principle. Antibiotics should be adjuncts to local and general therapy in otitis media and used only where indicated. Acute nonsuppurative otitis media does not require antibiotic therapy but rather measures which restore aeration of the middle ear and permit drainage of the secretions. These measures include myringotomy and treatment directed to the cause of the tubal obstruction. The incidence of hearing loss from serous effusion of the middle ear is higher than ever.

To be effective in the treatment of otitis media, antibiotic therapy should be combined with the use of oral and topical decongestants. Antibiotics must be given in adequate dosage and over a sufficiently long period of time. The use of antibiotic drugs without myringotomy may result in sterile exudate remaining in the middle ear, predisposing toward loss of hearing. Attention must also be directed towards the presence of large adenoids, nasal allergy, or chronic sinusitis. The patient must be observed not only until fever and discharge have ceased but until the ear drum has returned to its normal appearance and position and the hearing has returned to normal. Irradiation of the nasopharynx may be indicated in selected cases in order to restore normal hearing. If a hearing loss is to be discovered in the early period, when it is most amenable to treatment, the physician treating otitis media must be constantly alert to its presence.

Jennes, Waterbury, Conn.

Piromen Therapy in Otitis Externa. R. C. Welsh, Eye, Ear, Nose & Throat Month. **32**:585 (Oct.) 1953.

In a preliminary report on the use of Piromen, a Pseudomonas polysaccharide, in the treatment of 21 cases of acute otitis externa, Welsh claims a reduction in the duration of the disease. With local treatment alone, the symptoms usually lasted from 6 to 10 days. With the addition of Piromen, there usually was relief from symptoms within four days.

Jennes, Waterbury, Conn.

Evaluation of Hearing Loss in Drop Forge Workers. Meyers S. Fox, Laryngoscope **63**:960 (Oct.) 1953.

The hearing loss experienced by 64 drop forge workers is analyzed. Taken into consideration are the loudness and range of noise, its duration (length of exposure), age of the worker, and condition of the worker's ears when first subjected to the noise. Failure to obtain hearing

levels at this time (i. e., when first employed) was embarrassing to the employer. All losses were of the perceptive type, often most marked at 4096 d.v. The speech perception sometimes was not similar to the audiometric findings. Particularly because of the medicolegal aspects of the problem, a better way will have to be found to evaluate such hearing losses.

HITSCHLER, Philadelphia.

FUNCTIONAL AND HISTOLOGICAL FINDINGS AFTER OBLITERATION OF THE PERIOTIC DUCT AND ENDOLYMPHATIC SAC IN SOUND-CONDITIONED CATS. HAROLD F. SCHULKNECHT and ROBERTS S. KIMURA, Laryngoscope **63**:1170 (Dec.) 1953.

Destruction of the endolymphatic sac failed to create cochlear dysfunction or histological changes in the membranous labyrinth. Surgical blockage of the periotic duct (the channel of loose connective tissue and contained fluid which lies within the cochlear aqueduct) was associated with a moderate rise of Reissner's membrane in two ears, but this relationship is not a certain one for several reasons. The operative technique and histopathologic findings are explained.

HITSCHLER, Philadelphia.

TREATMENT OF THE CHRONIC DISCHARGING EAR. JOSEPH A. SULLIVAN, New York J. Med. **53**:2486 (Nov. 1) 1953.

Treatment of the chronic discharging ear was undertaken in the Department of Otolaryngology at the University of Toronto. The aims were (1) to eliminate intracranial complications, (2) to obtain a dry ear, and (3) to preserve and, if possible, improve the hearing. Six types of chronicity were observed. 1. The first type is drum membrane involvement with only a central perforation. If the ear can be dried and the perforation closed, hearing will improve. 2. In the second type the infection has destroyed the lining mucosa, part of the drum membrane, and the annulus. Cholesteatoma may be present, with saprophytic organisms in the attic. If other treatment fails, a modified radical mastoid operation is in order. 3. A focus of dead bone persists and discharge is scant, serous, and foul. Perforation may be anywhere, but there is no cholesteatoma. Surgery is required. 4. In the fourth type the onset is insidious and without acute symptoms; there is perforation of Shrapnell's membrane only. Also found are Eustachian-tube blockage, cholesteatoma with foul discharge, and erosion of the tympanic cavity. This type of cholesteatoma is called "primary acquired cholesteatoma." If hearing is below 35 db., the modified radical mastoidectomy may be tried. 5. The allergic type is associated with nasal pathology (rhinitis, sinusitis). This is accompanied by hypersecretion, an odorless mucoid discharge, and hypersensitivity to bacterial proteins. The nasal condition must be cured before the aural discharge will cease. 6. In the sixth type chronic discharge is due to the tubercle bacillus, either the human or bovine organism. These cases are rarely seen. Treatment of chronic otitis consists of (1) thorough cleansing without producing trauma, (2) maintenance of a dry cavity, and (3) the use of sulfonamides, penicillin, and streptomycin powder should be withheld for postoperative cavities. The pH is kept acid or is alternated with alkaline, according to the type of organisms present. Bacillus pyocyaneus is usually found. A modified radical mastoidectomy is the operation of choice to conserve or improve hearing. If this fails, a complete radical must be done, with removal of the annulus and closure of the tube and with a skin graft, if desirable in the given case.

I. W. VOORHEES, New York.

DIHYDROSTREPTOMYCIN AND DEAFNESS. JAMES K. LEES and DONALD M. MARKLE, New York J. Med. **53**:2997 (Dec. 15) 1953.

Lee and Markle work in the Department of Otolaryngology, College of Physicians and Surgeons, Columbia University, but this study was carried out at the Summit Park Sanatorium, Pomona, N. Y. Both streptomycin and its analogue, dihydrostreptomycin, affect the auditory nerve adversely, and deafness therefrom is irreversible. The early dosage of 1 to 2 gm. daily was too strong. Now the results are just as good with 1 gm. thrice weekly. Merck's product was used. Tests were carried out in an 18 by 20-ft. room, heavily draped and carpeted, with an overall noise index of 34 to 46 db., using a Western Electric 6 BP audiometer. Sixty patients

were screened, of whom 53 were taken for complete studies. All had received 1 gm. of dihydrostreptomycin two or three times a week. A 20-db. intensity level was chosen. The Spearman rank-difference method was adopted for correlative purposes. A table is given to show the air-conduction threshold of 31 patients with hearing below the 20-db. screening level. It was found that when 1 gm. of dihydrostreptomycin sulfate was administered two or three times a week, the incidence of deafness was remarkably reduced and involved the higher range, usually beyond the conversation area. This would seem to indicate that a relatively low dosage of dihydrostreptomycin can be used almost with impunity in treating tuberculosis, with little loss of conversational hearing, if any.

I. W. VOORHEES, New York.

FATIGUE AND ADAPTATION OF THE CUPULAR MECHANISM OF THE HUMAN HORIZONTAL SEMICIRCULAR CANAL: AN EXPERIMENTAL INVESTIGATION. C. S. HALLPIKE and J. D. HOOD, Proc. Roy. Soc., London, s.B **141**:542 (Sept. 9) 1953.

The authors present experimental work as an added contribution to their previous work on the physiology of the cupula in the horizontal semicircular canal. In this study special attention is given to the factors of fatigue and adaptation of the cupula to prolonged stimulation. The duration of the after-sensation (vertigo) following rotation is used as a measure of the reaction. A special chair designed by the authors is used, by means of which both speed and acceleration can be accurately controlled. The treatment of the physiological mechanism of the cupula follows that described by van Egmond, Groen, and Jonkees, whose findings the authors have confirmed in previous communcations. In these they were able to show that the duration of the after-sensation following turning can be related to the time taken by the cupula to return to its position of rest after it has been deflected by turning. Because of some irregularities, which seemed likely to be attributable to fatigue or adaptation, in the duration of the after-sensation in their previous experiments, these additional experiments were employed to further elucidate the physiological nature of the cupular response.

The authors discuss the subject of fatigue and adaptation, which is part of their objective in these experiments, so as to apply these in relation to the cupula. Under the heading of "Fatigue Effects," it is stated, are included two phenomena, adaptation and "true fatigue." In order to facilitate the discussion, the noncommital term response decline (R.D.) is introduced to denote any loss of sensitivity arising from fatigue or adaptation. Fatigue is characterized by a R.D. which develops slowly. It increases with stimulus intensity and progresses indefinitely with time. Its recovery is slow. Adaptation is characterized by a R.D. which develops rapidly at first and thereafter more slowly, finally reaching a constant and fully adapted level. A comparison is made with stretch receptors in muscle and with certain findings in cochlear adaptation reported by the same authors. The latter findings are based on the electrophysiological investigation of the fall in the action current response of the mammalian cochlear nerve to sustained pure-tone stimulation.

Briefly, the authors' procedure in the present experiment is to give a standard test stimulus of 4 degrees per second per second for six seconds, followed by a period of rotation at constant velocity, in effect, a rest period during which the cupula returns to its rest position.

After this a prolonged stimulus of 2 degrees per second per second for 75 seconds, designed to bring about an R.D., is then applied, and this in turn is followed after a rest interval by a further standard test stimulus. The occurrence of a R.D. was demonstrated by a considerable shortening of the after-sensation duration of the second test stimulus with respect to the first. The prolonged test is applied in both directions, thus tesing the function of the cupula away from and towards the ampulla. The prolonged test stimulus has been designed to eliminate interfering effects, such as centrifugal force, tactile sensations, and air flow. In practice, after the measurement of the after-sensation duration of the first test stimulus of 4 degrees per second per second, with rotation of the chair to the right, it was gradually reaccelerated from rest to the left at 2 degrees per second per second over a period of 75 seconds, the terminal velocity being 150 degrees per second. The velocity was maintained constant for 60 seconds in order to allow the cupula to return to its position of zero deflection and to recover from any adaptation or fatigue. It was then decelerated to rest at 2 degrees per second per second over a period of 75 seconds, this constituting the prolonged stimulus designed to give a R.D. After a further

rest interval of 40 seconds, to allow the cupula to return to zero deflection, the second test stimulus was applied as before, i. e., acceleration to the right at 4 degrees per second per second. The duration of the after-sensation was then found to be reduced, evidence of an impaired response due to prolonged stimulation. When greater prolonged stimuli were employed, such as 4, 5, and 6 degrees per second per second for 30 seconds, these greater stimuli did not significantly alter the R.D. of the test stimulus compared with that brought about by a prolonged stimulus of lesser intensity. This result is in agreement with the authors' previous investigations on the cochlea and is adduced as evidence that the R.D. is due to adaptation of the cupula end-organ.

When the test stimulus is applied in such a direction that the cupular deflection arising therefrom is opposite in direction to that produced by the prolonged stimulus, no R.D. was observed.

This suggests a directional specificity of the cupula, i. e., prolonged cupular deflection in one direction diminishes its subsequent response to deflections in the same but not in the opposite direction.

The authors also describe these tests on three patients with surgically destroyed labyrinths on one side. One case is described in detail. These also exhibit the phenomena of directional specificity, confirming the existence of an independent mechanism within each cupula capable of subserving the entire pattern of cupular adaptation. That the reactions described are due to adaptation rather than fatigue are discussed by the authors at length, with particular reference to the adapted state described by Matthews. The authors further discuss their findings in relation to Ewald's experiment and conclude that the cupula is a bidirectional signaling mechanism. Also there is a discussion in the light of their experimental work with electrical responses (action potentials). The structure of the crista is also discussed, with reference to the descriptions by Cajal and Lorente de Nó, as having two types of fibers—colossal and fine. This is discussed in the light of their present experiment, and a new theory is propounded on the nervous mechanim of the cupula. The latter discussions cannot be easily contracted and must be studied in the original of the authors' paper.

FROM THE AUTHORS' SUMMARY.

SUBJECTIVE TINNITUS OR TINNITUS AURIUM. R. S. VENTERS, Proc. Roy. Soc. Med. **46**:825 (Oct.) 1953.

Venters' address was given before the Section of Otology of the Royal Society of Medicine March 6, 1953, Air Vice-Marshal E. D. D. Dickson presiding. He gives a differential etiology in 254 cases and mentions infectious middle-ear disease, degenerative deafness, otosclerosis, trauma, Ménière's disease, syphilis, eighth-nerve tumors, anemia, psychogenic causes, and some five unclassified causes. He states that "cochlear or neural involvement determines the presence or absence of tinnitus, ten, twenty or thirty years later." Special attention is paid to degenerative deafness in 20% of his total cases. Emotional distress, worry, and fatigue are often associated with noises in the ears. Both mental and physical dysfunction are seen. In summary, the incidence of tinnitus may or may not be concerned with age groups. Middle-ear disease is the commonest primary cause. Neural disorders may involve the cochlea, giving rise to tinnitus. There is no known effective treatment.

I. W. VOORHEES, New York.

DETECTION AND RE-EDUCATION OF THE DEAF CHILD AS CARRIED OUT IN SOUTHWEST OF FRANCE. JEAN DESPONS, MICHAEL PORTMANN, CLAUDINE PORTMANN, and ODETTE TAILLEFER, Presse méd., Supp. 81, p. 3 (Dec. 20) 1952.

The problem of infantile deafness is studied in the southwest of France through three stages: detection, diagnosis and exact evaluation of the deafness, and direction to be given to the audiphonic reeducation.

Detection at preschool age is chiefly effected by the attentive parents or physicians of general medicine, infantile psychiatry, and phonology. Any delayed speaking should necessitate an audiometric examination. An important point is the school medical inspection with its systematic tests and special audiometric and otorhinolaryngological examination of any child who is backward, lazy, defectively speaking, etc.

Evaluation of deafness is made in an especially well-fitted laboratory (telephonometric room), where the following tests are performed by specialists of infantile audiometry: tonal and vocal audiometry, picture test, reaction of the name call, bells test, psychogalvanometry (Laboratory of Audiology, Service O. R. L., Hospital Saint-André, Bordeaux).

The direction to be given to the reeducation is secured by comparing the audiometric, psychiatric, and familial inquiries. When the children show a normal mental level they are directed, according to the degree of deafness, either to the school board for deaf and dumb or to the reeducation courses at the Phono-Audiology Center (Service O. R. L., Hospital Saint-André, Bordeaux) with electroacoustic amplification for minor half-deaf and major half-deaf. The latter will be reeducated by creating a phonologic conscience through such means as auditory training by amplification, visual training (labial reading, oscilloscopy), reading and articulation, etc. Such treatments may be started as early as 4 to 5 years of age; they are preceded by a stay in the "Jardin d'enfants" where the auditory paths and centers are first educated by amplification of the usual noises.

In conclusion, the authors point out that an audiometric and psychiatric investigation should be carefully carried out before making the grave decision on the choice of the establishment suitable for the child.

Such a laboratory, as available in Bordeaux, constitutes a sorting center where the direction to be given to the phonologic reeducation of deaf children is determined in a most rational and efficient way.

FROM THE AUTHORS' SUMMARY [A. M. A. AM. J. DIS. CHILD.].

INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN. H. VALLADARES and R. POBLETE G., Rev. otorrinolaring. **12**:1 (April) 1952.

Valladares and Poblete discuss a series of 109 cases of septic intracranial complications. These include parenchymatous abscess, subdural abscess, epidural abscess, encephalitis, thrombophlebitis, and meningitis. Of this group 34 were directly related to either otitis media or mastoiditis. Of the entire group of 109 cases, there was a total mortality of 44 patients. However, the authors split these mortality statistics into three periods, due to both changes in technique and treatment. From 1940 to 1945 in a series of 46 complications, 24, or 52.1%, died. From 1946 to 1948, thirteen, 43.3%, of a series of 30 patients died. From 1949 to August, 1951, there were a series of 33 patients, in which only 7, or 21.2%, died.

Their approach to treatment is first the acute stage, to permit formation of a firm capsule. This usually takes from three to four weeks. This is provided that the symptoms are not so severe to indicate extreme emergency measures. The second phase is making an accurate diagnosis, so that the extent of the operation can be minimized and drainage can be established in the most convenient and simple manner. They employ three methods of diagnosis. 1. A ventriculogram, either by lumbar puncture or suboccipital puncture. This, as a rule, is done very cautiously, since there is danger of a rupture of the abscess into the fourth ventricle. Because of this risk they are extremely cautious in its use. 2. Arteriography. This is an injection of a contrast medium into the arterial tree. This diagnostic means has the advantage of not changing the intracranial pressure and is extremely valuable in diagnosing lesions in the temporal, frontal, and parietal areas. Valladares and Poblete prefer this method of diagnosis to a ventriculogram when they suspect the frontal or temporal lesions. 3. Electroencephalogram. This is extremely valuable in localizing lesions in the anterior fossa. The method is innocuous, and it is one that is used repeatedly when the lesion in this area is suspected. The surgical treatment is usually puncture drainage. The authors do not advocate the excision of the capsule, since frequently this would involve a greater amount of vital tissue, and postoperative complications are by far more common.

PERSKY, Philadelphia.

RELATION BETWEEN HEARING AND THE DEGREE OF MIDDLE-EAR PNEUMATIZATION. S. HARAGUCHI, J. Oto-Rhino-Laryng. Soc. Japan **56**:595, 1953.

Haraguchi classified roentgenologically middle-ear pneumatization with regard to its degree of development into three groups: (*a*) overdeveloped, (*b*) well-developed, and (*c*) undeveloped. An audiometric examination was made on 18 cases with overdeveloped pneumatization, 22

cases with bilaterally undeveloped pneumatization, and 16 cases with a unilaterally undeveloped air-cell system, selected from 206 healthy youths. There was found no remarkable difference in the threshold for air-conducted sound between the groups with overdeveloped and undeveloped pneumatization. The threshold for bone-conduction sound in the latter was about 5 db. lower than that in the former, using a frequency between 256 and 2048 cps. In the well-developed cases the hearing gain for bone-conducted sound with the external auditory canal closed was 10 to 20 db. for low frequencies. In the undeveloped cases it was only about 5 db. From these results Haraguchi discussed the part played by the air-cell system in hearing.

HARA, Los Angeles.

Pharynx

CONTROVERSIAL PROBLEMS IN ADENOTONSILLECTOMY. JOHN W. ARNOLD, California Med. **78**:444 (May) 1953.

It is doubtful that the tonsils or adenoids contribute sufficiently to immunity or to hematopoiesis to warrant withholding adenotonsillectomy when there is need for the operation. Focal infection, rheumatic fever, and allergic disease must be evaluated in the individual case. A seldom-discussed reason for operation is the prophylaxis or treatment of malformation of the nose, sinuses, mouth, and jaw. Well-defined indications for adenotonsillectomy include frequent occurrence of infection, peritonsillar abscess, cervical lymph-node disease believed caused by tonsillar infection, otitis media, and hypertrophy sufficient to embarrass swallowing or breathing. The operation may also be indicated in certain cases of impaired hearing, halitosis, or anorexia, and sometimes for carriers of diphtheria.

Psychic trauma can be obviated by proper preparation of a child for the operation he is to undergo.

There is a good deal of evidence of relationship between recent adenotonsillectomy and infection with poliomyelitis—and a good deal of evidence to the contrary. Nationwide rules cannot be established on the basis of the evidence presented thus far. Since in many cases it is unwise to put off adenotonsillectomy, each case in each community in each season must be evaluated separately.

PRYOR, Redwood City, Calif. [A. M. A. AM. J. DIS. CHILD.].

STATISTICAL STUDY OF POSTOPERATIVE TONSIL AND ADENOID HEMORRHAGE. RICHARD J. BELLUCCI, New York J. Med. **53**:2495 (Nov.) 1953.

Bellucci made a statistical study at the Manhattan Eye, Ear and Throat Hospital of all cases of adenotonsillectomy admitted for ward service since 1948. Almost 20,000 (exactly 19,754) charts were reviewed, of which there were 930 cases of primary hemorrhage and 208 cases of secondary hemorrhage. Weather conditions and the nationality, age, race, sex, and coloring of the patient seemed to have no bearing on the incidence of bleeding. History of a recent upper respiratory infection was not reflected in the expected increase of postoperative bleeding. But a higher incidence was recorded in the presence of carious teeth, mucopurulent discharge in the nose and throat, and large, tender cervical lymph nodes. Tonsils which contained infected debris and those which were adherent to the fossae seemed to have more frequent hemorrhage after surgery. So-called "primary hemorrhages" were more often located in the nasopharynx after adenoid removal. Secondary bleeding was more often located in the tonsillar fossae. Surgical skill and patience in controlling bleeding are the sine qua non of successful adenoid and tonsil surgery and postoperative results. Ninety-six percent of the patients have shown general physical improvement after these operations. Postoperative bleeding can be reduced materially by removing or relieving all upper respiratory infections preceding surgery. Careful hemostasis before the patient leaves the operating table is imperative.

I. W. VOORHEES, New York.

SIALOLITHIASIS. BERNARD J. FICARRA, New York J. Med. **53**:3039 (Dec. 15) 1953.

Ficarra states that the formation of stone in the salivary glands is relatively infrequent compared with stone formation in the bile ducts and elsewhere. A physician may see only one or two such conditions in a lifetime of practice. Three causes are set down; namely, infection,

stagnation, and concentration. The presence of any one of these factors is capable of activating the others, and it may take months or years before acute symptoms bring a patient to his physician. Then, there is commonly pain due to distention of the gland and formation of pus. The stone may be removed from within the mouth, but in the case reported by Ficarra, external operation was necessary for removal of the left submaxillary gland in toto. The pathologist reported a dense accumulation of lymphocytes within the interstitial connective tissue, with occasional dilatation of the ducts. Chronic inflammatory fibrosis was present. Recovery was uneventful.

I. W. VOORHEES, New York.

THE TONSIL AND ADENOID PROBLEM. G. E. TREMBLE, Canad. M. A. J. **68**:9 (Jan.) 1953.

The history of tonsillectomy is reviewed briefly, and indications for the operation are discussed. The author stressed that point that operation should depend at least 50% on the history, and points out that the appearance of the tonsils is by no means an adequate basis for determining the need for operation. He feels that adenoidectomy alone may be of benefit in repeated colds, ear infections, or nasal blockage. In the absence of frequent sore throats or cervical adenitis the tonsils should be left.

MCGUIGAN, Evanston, Ill. [A. M. A. AM. J. DIS. CHILD.].

DISCUSSION ON THE EARLY DIAGNOSIS OF NASOPHARYNGEAL CARCINOMA. WILLIAM GOODDY, Proc. Roy. Soc. Med. **46**:817 (Oct.) 1953.

This paper was read on March 6, 1953, before the Section of Laryngology, President J. P. Stewart presiding. Gooddy is a neurologist, whose interest in nasopharyngeal carcinoma stems from his study of patients with facial pain. Symptomatology aids in making an early diagnosis. One finds glandular, otological, rhinological, and neurological symptoms. Those of dental origin are also mentioned. Trigeminal neuritis gives rather continuous pain without motor involvement to any extent, save for the sixth nerve, with paralysis of the external rectus. Double vision may occur with blurring. Nystagmus is rare. One should watch for sudden electric stabs of pain, often unilateral only, with intermittent freedom. Any slight touch, cold wind, etc., may produce an attack. The so-called "trigger points" are at angles of the mouth or vestibuli nasi. Migraine may becloud the symptoms of neoplasm. Aneurysm of circle of Willis gives rise to throbbing pains. "Symptoms without objective signs must be regarded always with the greatest suspicion." The most important symptom is pain in the face. In all such cases a complete rhinological examination, with special attention to the nasopharynx, is imperative.

I. W. VOORHEES, New York.

Larynx

PROBLEMS IN DIRECT LARYNGOSCOPY. F. S. FORMAN, Eye, Ear, Nose & Throat Month. **32**:79 (Feb.) 1953.

Direct laryngoscopy is indicated when complete, satisfactory examination of the larynx for diagnostic purposes is not possible with mirror examination and when surgical intervention is necessary. Psychologic preparation, morphine and scopolamine preoperative medication, and careful local anesthetization are highly essential prerequisites. Cocaine, in 5% solution, is the most satisfactory topical anesthetic agent. It is applied in the following way: The hypopharynx is sprayed with the 5% solution. After two and one-half minutes, the larynx is exposed by indirect laryngoscopy, and 4 to 5 drops of the 5% solution are applied onto the vocal cords with a Luken's syringe. This is repeated twice at intervals of five minutes. It is of considerable importance for the surgeon to apply the anesthesia himself, because he is responsible for its adequacy. The reactions occurring from cocaine are largely due to the injudicial use of high concentrations and perilously large doses. It is suggested that a relatively safe maximum of 5 cc. of a 5% solution be employed. The secret of avoiding reactions, or at least minimizing them, is to wait for at least five minutes between applications of the anesthetic solution. There is no place for hurry in producing topical anesthesia in the larynx with cocaine or its substitutes.

While local anesthesia is usually adequate, there are times when it fails. General anesthesia is indicated in the apprehensive patient who cannot be psychologically prepared, in the patient whose anatomical structure makes exposure difficult, and in those in whom prolonged exposure or very delicate work is necessary. For general anesthesia, thiopental sodium (Pentothal)-curare anesthesia has proved quite satisfactory.

The most commonly overlooked factor in direct laryngoscopies is the position of the patient. If the position is correct, many of the difficulties are readily overcome. Manual head-holding is ideal. If this is not available, or if laryngoscopy is infrequently done, mechanical head holders are serviceable. Although most laryngoscopies can be done with the Jackson laryngoscope, there are some in which exposure of the anterior commissure is difficult. Modifications of the Jackson laryngoscope by Hollinger and Andrews will aid in these difficult cases. The Roberts laryngoscope incorporates most all features of other laryngoscopes to facilitate complete exposure of the larynx.

JENNES, Waterbury, Conn.

BILATERAL ABDUCTOR VOCAL CORD PARALYSIS. W. C. THORNELL, Eye, Ear, Nose & Throat. Month. **32**:305 (June) 1953.

In patients who have bilateral abductor vocal cord paralysis and who have an inadequate airway or who have required tracheotomy and who have shown no improvement within six months following the onset of this condition, an attempt to correct or improve the condition should be considered. There are two objectives of the surgical procedures: first, the establishment of an adequate airway and, second, the preservation of the voice. An adequate airway requires separation of the vocal cords, whereas a normal voice requires close approximation of the vocal cords. The problem in the management of these patients is to move one paralyzed vocal cord laterally far enough to create an adequate airway without moving it so far laterally as to destroy the voice. Of the two objectives, establishment of an adequate airway is the more important.

Thornell has previously presented a technique of intralaryngeal arytenoidectomy, to which he has added subsequently a few minor modifications. The removal of the arytenoid cartilage by means of the intralaryngeal approach is carried out with the larynx exposed to direct inspection through the mouth by means of a self-retaining laryngoscope. Thornell describes in detail the steps in the surgical techniques.

Continued reports of successful end-results are being received from surgeons using the approach described. Of 160 cases reported by more than 25 laryngologists, successful end-results have been obtained in 147 patients. Of the remaining 13, two refused further surgery. Of the 11 patients who had arytenoidectomy carried out on the opposite side, results were successful in 8. The end-results obtained from arytenoidectomy in the treatment of bilateral abductor vocal cord paralysis by means of the intralaryngeal approach compares favorably with the end-results reported by other surgeons using different methods.

JENNES, Waterbury, Conn.

MANAGEMENT OF CANCER OF THE LARYNX. F. C. ORMEROD, J. Laryng. & Otol. **68**:1 (Jan.) 1954.

In delivering the Semon lecture, Ormerod reviews the historical data relative to laryngology and the origin of the Semon lecture. Increased experience and knowledge of laryngeal carcinoma warrants a change in the division and classification of malignant tumors of the larynx into the glottic, subglottic, and supraglottic. Irradiation is discussed and advocated, and while the response of anaplastic carcinoma to radiotherapy is good, the results obtained with the more differentiated cell tumors is far superior. The prognosis of carcinoma arising in various regions of the larynx is well presented and discussed, and a warning is sounded on the dangers of transmitting cancer cells to healthy portions of the respiratory tract on instruments, hands, gloves, or intratracheal anesthetic tubes. The indications for thyrotomy, laryngectomy, and block dissections of the neck are discussed, and preoperative as well as postoperative speech training for laryngectomized patients is advocated.

LEJEUNE, New Orleans.

BILATERAL ABDUCTOR PARALYSIS OF VOCAL CORDS DURING LOCAL TONSILLECTOMY: REPORT OF A CASE AND ITS MANAGEMENT. GEORGE S. RICHARDSON, Laryngoscope **63**:1197 (Dec.) 1953.

Bilateral abductor paralysis of the vocal cords occurred during a local tonsillectomy. Novol (butethamine [Monocaine] hydrochloride, 1.5%, with epinephrine, 1: 100,000) was used as the anesthetic. There were no other untoward effects. The paralysis was relieved by the injection of 10 mg. of diphenhydramine (Benadryl) hydrochloride intravenously.

HITSCHLER, Philadelphia.

Nose

CHRONIC MAXILLARY SINUSITIS IN CHILDHOOD. G. M. H. VEENEKLAAS, Presse méd. **58**:479 (April 26) 1950.

Maxillary sinusitis occurs more frequently in childhood than is suspected and may lead to grave impairment of the general physical condition. Sinusitis may be brought about by obstruction from allergic reaction, by infections of the tonsils and adenoids, as a complication of contagious disease, as a result of underwater swimming, or from a cold damp climate. Sinusitis may lead to infection of the neighboring structures of the nasopharynx and to bronchitis. It may affect the development of asthmatic attacks or be the forerunner of endocarditis, arthritis, or nephritis.

Roentgenograms, properly taken, will establish the diagnosis. Treatment should start with elimination of the inciting cause. If shrinkage of the affected mucous membranes by use of ephedrine nose drops does not clear the sinuses, penicillin should be used. If this is not successful, lavage of the sinus may be tried. Sometimes this procedure has to be repeated several times in order to effect a cure. Occasionally a very stubborn case may yield to the use of autogenous vaccine, or more radical surgery may cure the condition.

DAFFINEE, Melrose, Mass. [A. M. A. AM. J. DIS. CHILD.].

LUPUS OF THE NOSE AND STREPTOMYCIN. F. CORDARO, Rev. otorrinolaring. **12**:21 (April) 1952.

Cordaro presents four patients (13, 14, 30, and 48 years of age) with lupus of the nose. These were treated with dihydrostreptomycin in a dosage of 1 gm. daily for a period of seven to eight weeks. The results were extremely brilliant. All lesions were completely cured, and there was very little influence on either the vestibular or cochlear divisions of the eighth nerve. The mutilating treatment with either caustics or electrocoagulations has been definitely abandoned. Any other evidences of tuberculous toxemia were either favorably influenced or completely suppressed in the course of the treatment.

PERSKY, Philadelphia.

EXPERIMENTAL PATHOLOGICAL STUDY OF NASAL MUCOUS MEMBRANE. K. HARA, J. Oto-Rhino-Laryng. Soc. Japan **56**:463, 1953.

By injecting such pigments as ink of squid and drawing ink and metallic poisons, e. g., bismuth, silver, and mercury, into the peritoneal cavity of adult mice, I histologically investigated the possibility of these pigments and metallic granules being eliminated into the nasal cavity.

Pathological changes of the nasal mucous membrane in the presence of systemic infection was investigated histologically by injecting such objects as diphtheria toxin and staphylococcic toxoid and animal poisons like hepatotoxin in addition to metallic poisons into the peritoneal cavity of animals. The results were as follows:

On the nasal mucous membrane acute inflammatory or regressive changes were observed, though the degree of the changes differed from case to case; and on the respiratory mucous membrane marked inflammatory changes of the catarrhal or suppurative type were present. On the

olfactory mucous membrane, a regressive picture of degeneration, collapse, and destruction was conspicuous. All these changes were most marked in the nasal cavity and in a much less degree in the accessory sinuses and Jacobson's organs.

The pathological changes mentioned above were marked in cases of metallic poisons, especially mercury and bismuth. They were generally slight in cases of bacterial toxins, except diphtheria toxin, which induced intensive changes. Inflammatory changes were hardly to be found when hepatotoxin, an animal poison, was applied.

FROM THE AUTHOR'S SUMMARY.

Miscellaneous

TREATMENT OF ALLERGIC DISEASES WITH PIROMEN. A. WOLF and H. S. HARTLEY, Eye, Ear, Nose & Throat Month. **32**:261 (May) 1953.

Piromen is described as a sterile, nonprotein, nonanaphylactogenic bacterial component derived from a Pseudomonas species. Wolf and Hartley call attention to the results of various investigators using this product in the treatment of allergic diseases. They report the results of studies of the effects of Piromen on skin tests in ragweed-sensitive patients and the clinical effects on patients with nasal allergies. Ragweed-sensitive patients were selected for titration of skin sensitivity before and after the subcutaneous administration of 0.3 cc. of a solution containing 10 γ per cc. of Piromen three times a week for 30 days. No changes in the size of the intracutaneous wheal were obtained.

Patients with nasal allergies were selected for clinical evaluation of Piromen. The symptoms of the patients were moderately severe. Routine injections of inhalants were discontinued, and each patient received 0.3 cc. of a solution containing 3 γ of Piromen or a Piromen placebo three times a week for 30 days. After this period, a clinical evaluation was made. No change in the skin sensitivity in the ragweed-sensitive patient could be demonstrated. The clinical response of the patients with nasal allergies was disappointing. The number of patients who improved and those who failed to improve were about the same in the group receiving 3 γ of Piromen and the group receiving placebo injections, respectively.

JENNES, Waterbury, Conn.

HAS OTOLARYNGOLOGY A FUTURE? AN EDITORIAL, Eye, Ear, Nose & Throat Month. **32**:451 (Aug.) 1953.

The future of otolaryngology has been the subject of several articles and editorials during the past few years. Younger doctors have felt that otolaryngology is now strictly a nonsurgical specialty. This is, of course, remote from the truth. The range in surgery in otolaryngology is broader than is generally realized. One is no longer a specialist because he can perform a tonsillectomy and a septal resection. There have been new advances, both surgical and nonsurgical, in modern otolaryngology.

Among the surgical advances are the correction of nasal deformities, the removal of involved salivary glands, the treatment of pathologic processes of the ear, larynx, and neck, and endoscopy. The extirpation of neoplasms of the head and neck and plastic reconstruction of these parts are also functions of the well-trained otolaryngologist. The more important medical advances include nasal allergy and audiology.

The writer believes that otolaryngology quite definitely has a future. For each physician, otolaryngology is as broad as his training, skill, and experience will permit. The shrinking number of younger doctors entering the specialty has made it an especially desirable branch. Physicians contemplating a specialty should not permit themselves to be misled. The future of otolaryngologic surgery is bright, and medical aspects have been extended. The future of otolaryngology rests with the individual doctor. If he emerges from his training adequately qualified, his future will be a bright and successful one.

JENNES, Waterbury, Conn.

THE DIAGNOSIS AND TREATMENT OF ALLERGIC RHINITIS. WALTER E. OWENS, Illinois M. J. **103**:283 (May) 1953.

Allergy is an important phase of rhinology. Histamine release is responsible for allergic symptoms. Adequate history, physical examination, cytology of nasal secretions, and skin tests are necessary for a diagnosis of allergy. Seasonal allergic rhinitis is due to pollen sensitivity. Small optimum dosage technique as recommended by Hansel is used. Careful use of antihistaminic and sympathomimetic drugs are of value in treatment.

FROM THE AUTHOR'S SUMMARY [A. M. A. AM. J. DIS. CHILD.].

ACUTE ASTHMATIC EPISODES IN CHILDREN CAUSED BY UPPER RESPIRATORY BACTERIA DURING COLDS, WITH AND WITHOUT BACTERIAL SENSITIZATION. FRANKLIN A. STEVENS, J. Allergy **24**:221 (May) 1953.

Asthma may occur with colds in children if they are focally hypersensitive to the bacteria causing the second stage of the cold. In the purely infectional type of asthma, attacks occur only with colds during the winter, not in summer. These patients require bacterial therapy. On the other hand, if there is a frank or even latent allergic bronchiolitis, although there is no bacterial sensitivity, the irritating and inflammatory effects of the infection alone will initiate an attack. Treatment of the underlying allergy is adequate in these patients. If there are several clinical sensitivities, it may be difficult to distinguish between these two mechanisms. If asthma occurs with colds during the summer, pollen sensitization should be suspected, and if the patients are sensitive to ragweed, asthma may occur with colds until early winter.

HOYER, Cincinnati [A. M. A. AM. J. DIS. CHILD.].

THE EYEBLINK RESPONSE AS A TEST FOR HEARING. ROBERT GALAMBOS, PHILIP E. ROSENBERG, and ARAM GLORIG, J. Speech & Hearing Disorders **18**:373 (Dec.) 1953.

The palpebral reflex of 53 subjects, 33 with hearing loss, was studied by recording the movements of one eyelid and stimulating both ears by means of a square-wave generator and earphones for producing clicks and a crystal phonograph cartridge and an oscilloscope for recording eyeblinks. In the normal subjects, eyeblink responses begin to occur at clicks around 50 db. above their threshold. Blinks occur more frequently with rising click intensity and appear every time at 130 db. above threshold. Subjects with hearing losses respond like normal subjects, except that higher intensities are necessary. This test does not clearly differentiate organic from nonorganic or conduction from "nerve-type" hearing losses.

PALMER, Wichita, Kan.

MALIGNANCIES OF THE EAR, NOSE, AND THROAT. DAVID W. BREWER, New York J. Med. **53**:2492 (Nov. 1) 1953.

Brewer works in the Department of Otolaryngology and Head and Neck Tumor Service at the State University of New York College of Medicine at Syracuse. He stresses the obligation of the E.N.T. man in making an early diagnosis of a tumor. Radon seeds, radium needles, and cobalt 60 have been used with increasingly good results. Electrosurgical coagulation is effective in many instances. A "group opinion" is of great value in diagnosing and treating such cases, but it is available more commonly in the larger than in the smaller cities. Two cases are reported: the first, a tumor of the middle ear and, second, cancer of the lower jaw and cheek. Brewer describes in detail all of the agencies used to effect a "cure." Both patients, having gone through the group routine, were still alive and seemingly well at the time of the report, May 7, 1953, before the Annual Meeting of the Medical Society of the State of New York held in Buffalo.

I. W. VOORHEES, New York.

THYROID CANCER. WILLIAM TREVOR, New York J. Med. **53**:2511 (Nov. 1) 1953.

Trevor concludes from his work at St. Clare's Hospital in New York that "Thyroid cancer is one of the least malignant of all cancers. If it is treated in time, the prognosis for five-year survival is excellent." Three signs should be looked for in making a diagnosis: Hoarseness is the first; it is due to infiltration of the recurrent laryngeal nerve. The second is fixation due to infiltration of the trachea and "ribbon muscles." Hardness is the third sign in this trilogy. It is

due to the outward extension of cancer tissue, which replaces the naturally soft tissue of the thyroid. Unfortunately, all of these are late signs of advanced malignancy. As for the age, the average age for cancer of the thyroid in women is 48.1 years and for men 52.8. Kennedy reported that in his cases carcinoma occurred in nontoxic nodular goiters. In children, almost half of all the goiters are carcinomatous. Goiter of all types is much more prevalent in females, but nodular goiter in males occurs in a greater ratio. "The solitary thyroid nodule carries a much greater incidence of malignancy than does the multinodular lesion. Lahey and Cole have emphasized this." In summary, it is well to remember the following: 1. Eighty percent of thyroid carcinomas orginate in preexisting adenomas. 2. Solitary thyroid adenomas carry a very high degree of malignancy. 3. In children a very high rate of malignancy is found in the thyroid nodules. 4. The ratio of thyroid carcinoma is three men to one woman. 5. All thyroid nodules should be thoroughly excised by subtotal thyroidectomy. Mortality should be nil when surgery is thoroughly and properly carried out. Hospitalization is only four days.

I. W. Voorhees, New York.

Effect of Aureomycin Treatment of Streptococcal Sore Throat on the Streptococcal Carrier State, the Immunologic Response of the Host, and the Incidence of Acute Rheumatic Fever. Harold H. House, George C. Eckhardt, Edward O. Hahn, Floyd W. Denny, Lewis W. Wannamaker, and Charles H. Rammelkamp, Pediatrics **12**:593 (Dec.) 1953.

A total of 1,009 patients with exudative tonsillitis or pharyngitis, predominantly streptococcal in etiology, was treated with chlortetracycline (Aureomycin) hydrochloride. A control group of 1,035 patients received no chlortetracycline.

An illness classified as definite or possible rheumatic fever subsequently developed in 20 patients who had received chlortetracycline and in 29 who had received no treatment. Data collected on rheumatic subjects indicated that when the interval between the onset of the observed attack of exudative tonsillitis or pharyngitis and the onset of rheumatic fever exceeded 35 days, an intervening streptococcal infection had occurred. Five patients in the treated group and 20 in the control group developed rheumatic fever within a 35-day interval. These data indicate that chlortetracycline therapy greatly reduces the subsequent occurrence of rheumatic fever.

Chlortetracycline therapy was also found to eradicate the streptococcus from the oropharynx in a large number of instances and to inhibit the formation of antistreptolysin. The degree of inhibition of antistreptolysin formation was shown to correlate with the eradication of the streptococci from the oropharynx. When bacteriologic relapse occurred following cessation of chlortetracycline therapy, the degree of inhibition of antistreptolysin production was low.

Chlortetracycline therapy of streptococcal infections was compared to penicillin therapy of such infections. It was shown that penicillin was more effective in eradicating streptococci, decreasing antistreptolysin formation, and preventing rheumatic fever. Penicillin, therefore, is the drug of choice.

From the Authors' Summary [A. M. A. Am. J. Dis. Child.].

Discussion on Hiatus Hernia and Esophagitis. J. B. Harman, Ronald Belsey, and H. D. Brown Kelly, Proc. Roy. Soc. Med. **46**:941 (Nov.) 1953.

The meeting of the Section of Laryngology of the Royal Society of Medicine on May 1, 1953, was presided over by President J. P. Stewart. Dr. Harman recited the symptoms of esophagitis, chief of which are chest pain and heartburn, caused by inflammation of the mucosa, not by spasm. Reflux of gastric acidity is a paramount incident.

Mr. Ronald Belsey's theme was the "Pathology and Treatment of Hiatus Hernia when Complicated by Esophagitis." He has studied some 250 cases, of which 200 were adults and 50 infants and children. Every case of hiatus hernia was esophagoscoped as a routine measure. X-ray evidence is insufficient. Advanced cases had more dysphagia than pain, some had no pain at all; but when increasing stenosis was seen, the difficulty of getting food through to the stomach was distressing. Biopsy specimens were taken from 40 patients. In all there was infiltration of round cells, with fibrosis extending to all coats of the esophagus. The mucosa was hypertrophic or replaced entirely, and mediastinal lymph nodes were enlarged. With stenosis, there was shortening of the lower third of the esophagus. The damage was frequently irrepair-

able, even by surgical resection. A curious feature as to stenosis is that it never becomes complete—there is always a tiny opening into the stomach. The esophagus is a kind of pump, and any interference with its neuromusculature may make difficult its function in working food downward. Endoscopic dilatation may be contraindicated in the presence of ulceration or lack of elasticity. Perforation is highly dangerous to life. The overall risk of dilatation may be even greater than surgical resection. Subtotal gastrectomy from below, with the idea of reducing gastric acidity, is contraindicated, since it rarely achieves the desired result and may complicate future resections of the cardiac end. "If the whole stomach is retained and the esophagus anastomosed to the fundus, the risk of recurring esophagitis at a higher level is considerable." Belsey prefers to resect the pepsin-secreting portion of the stomach and as much of the esophagus above the diseased area as he can manage. A sort of tube is then made of the greater curvature, and the anastomosis, when completely healed, is quite satisfactory. He states, "I have performed this operation on approximately 40 cases with satisfactory functional results and, as yet, no evidence of any recurring esophagitis at a higher level."

Mr. H. D. Brown Kelly's topic was "Management of Hiatus Hernia and Esophagitis." One must first establish an accurate diagnosis, excluding malignancy, and determine the presence or absence of ulcers. The endoscopy may disclose a dilated gullet with smooth walls and full of fluid. This is sucked out, and by moving the tube from side to side, one may estimate the amount of pathologic dilatation. Regurgitation of gastric juice may be encountered. At about 30 cm. from the upper teeth, one finds the gullet narrowing down to a rosette appearance at the cardiac end, through which the tube may pass without too much difficulty into the stomach, depending, of course, on the degree of constriction. Occasionally nothing will pass through, not even a fine bougie. In most cases, especially in adults, a sterile swab will show an acid reaction. In children with a short esophagus, the swab may show an alkaline reaction as far as the constriction, which becomes acid at the gastric introitus. As to treatment with silver nitrate, Kelly regards this as dangerous, because of the spread of the medication far beyond the desired area of attack and because of the greatly increased stimulation of the gland secretion. Any injury to the walls may cause increased obstruction. It is an aphorism that "those who live by the bougie eventually die of the bougie," but the risk of perforation is greater in an annular stenosis, due to cicatrices caused by corrosives. Much depends on the elasticity of the tissues and the pathology which has occurred throughout the preceding years.

I. W. VOORHEES, New York.

Books

Aeromedica Acta. Volume 1. Collected papers from the Foundation of the National Aeromedical Center, Soesterberg, Netherlands, 1952.

This is the first number of the Aeromedica Acta, which, it is hoped, will appear yearly. It contains papers from the "National Luchtvaartgeneeskundig Centrum" which are designated for this purpose by the Board of the Foundation. The papers are presented under three main headings: 1. "Some Observations on the Psychology of the Aviator," 2. "Motion Sickness," and 3. "Medical Examination of Flying Candidates and Causes for Rejection." All of these papers are provocative and should be of interest to those examining candidates for either military or commercial pilots.

One paper deals with the so-called monocular clues of distance perception, and the suggestion is made to provide every airport with a large and clearly visible geometrical figure of uniform size as a help for pilots in the judging of distances in unfamiliar surroundings. Another paper deals with the importance of oxymetry in aviation medicine; another concerns the data obtained from a simple attention test as compared with the qualifications resulting from psychological examination by means of a battery of tests. Another deals with the influence of intermittent light stimuli during various degrees of hypoxia on cerebral activity. Another discusses the rotatory test with small regulated stimuli, which has become known as cupulometry.

It is these latter papers which are of particular interest to the otolaryngologist.

Probleme der Ohrphysiologie und neue Losungsversuche.* By Max Kraus. Price, not given. Pp. 94, with 8 illustrations. Springer-Verlag, Mölkerbastei 5, Wien I, 1953.

Ear, Nose and Throat Diseases for Medical Students.* By William McKenzie, M.B., B.Chir. (Cantab.), F.R.C.S. (Engl.). Price, $5.50. Pp. 256, with 95 illustrations. E. & S. Livingstone, 16 and 17 Teviot Place, Edinburgh 1. Agents in the United States: Williams & Wilkins Company, Mt. Royal & Guilford Aves., Baltimore 2, 1953.

Dizziness, an Evaluation and Classification. By David Downs DeWeese, M.D. No. 176, American Lecture Series. Price, $2.75. Pp. 74, with 2 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1954.

Directory of Organizations and Individuals Active in the Fields of Deafness and Otological Research.* Compiled by Douglas Macfarlan, M.D., Director of Research Section on Audiology, Hospital of the University of Pennsylvania. Paper, 60 cents. Pp. 88. May be obtained from the Hearing Center, Hospital of the University of Pennsylvania, Philadelphia 4.

1954 Medical Progress: A Review of Medical Advances During 1953. Edited by Morris Fishbein, M.D. Price, $5.00. Pp. 331. The Blakiston Company, 575 Madison Ave., New York 22, 1954.

Physiological Acoustics.* By Ernest Glen Wever and Merle Lawrence. Price, $10.00. Pp. 410, with 9 plates plus numerous charts and diagrams. Princeton University Press, Princeton, N. J., 1954.

Essays on the Applied Physiology of the Nose.* By Arthur W. Proetz, A.B., M.D., Sc.D. Second edition. Price, $8.00. Pp. 424, with 130 illustrations. Annals Publishing Company, P. O. Box 1345, Central Station 1, St. Louis, 1953.

Year Book of the Eye, Ear, Nose and Throat (1953-1954 Year Book Series).* Edited by Derrick Vail, B.A., M.D., D.Oph. (Oxon.), F.A.C.S., F.R.C.S. (Hon.), and John R. Lindsay, M.D. Price, $6.00. Pp. 443, with 126 illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11, 1954.

* To be reviewed.

Directory of Otolaryngologic Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont.

International Bronchoesophagological Society

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Third International Congress of Bronchoesophagology.
Place: Lisbon, Portugal. Time: September or October, 1954.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Larynygology and Broncho-Esophagology

President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: United States of America. Time: 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary: Dr. Hugh A. R. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Walter H. Theobald, 307 N. Michigan Blvd., Chicago 1.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN BOARD OF OTOLARYNGOLOGY

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Statler Hotel, Boston.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3.
Place: Statler Hotel, Boston.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Boston.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
President-Elect: Dr. Kenneth M. Day, 121 University Place, Pittsburgh.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Statler Hotel, Boston.

SECTIONS:

Eastern.—Chairman: Dr. Daniel S. Cunning, 118 E. 53rd St., N. Y. 22.
Place: Hotel Syracuse, Syracuse, N. Y.
Southern.—Chairman: Dr. William C. Wolfe, 332 W. Broadway, Louisville 2.
Place: Andrew Jackson Hotel, Nashville, Tenn.
Middle.—Chairman: Dr. French K. Hansel, 634 N. Grand Blvd., St. Louis 3.
Place: The Drake, Chicago.
Western.—Chairman: Dr. Guy L. Boyden, 1735 N. Wheeler Ave., Portland 12, Ore.
Place: The Elks Club, Los Angeles.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Statler Hotel, Boston.

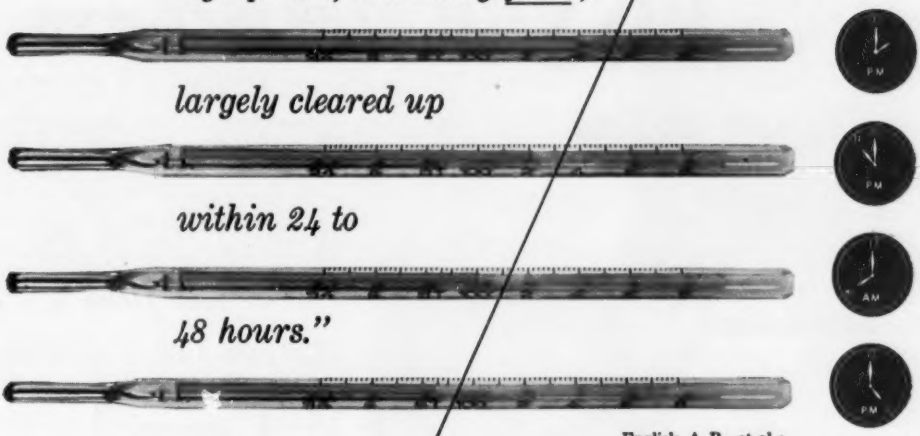
AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

President: Dr. Harry Neivert, 555 Park Ave., New York 21.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
President-Elect: Dr. Albert D. Reudemann, 1633 David Whitney Building, Detroit 26.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.

"*Symptoms, including fever,*



largely cleared up

within 24 to

48 hours."

English, A. R., et al.:
Antibiotics Annual (1953-1954),
New York, Medical
Encyclopedia, Inc., 1953, p. 70.

AIH *Tetracyn* hydrochloride

afebrile in hours c

Brand of tetracycline hydrochloride

a new broad-spectrum antibiotic of unexcelled tolerance

Simple, dramatic proof of the effectiveness of Tetracyn is offered by the characteristic rapid defervescence noted in the treatment of a wide range of susceptible infectious diseases. *Think of Tetracyn* whenever you take a temperature for an AIH response in Tetracyn-sensitive infections.

TETRACYN TABLETS
(*sugar coated*) 250 mg., 100 mg., 50 mg.

Rx PFIZER

BASIC PHARMACEUTICALS FOR NEEDS BASIC TO MEDICINE

536 Lake Shore Drive, Chicago 11, Illinois

New No. 41 Pocket Nebulizer by

DeVILBISS

Fills Long-felt Need

SUGGESTIONS FROM PHYSICIANS LEAD TO UNIQUE DEVELOPMENT

Spurred by suggestions from the medical profession, DeVilbiss has now perfected a pocket nebulizer which the asthmatic may carry with him at all times, ready to use at a moment's notice.

Leak proof, practically unbreakable. Provided with attractive carrying case. Weighs but an ounce and a half. Particle size and performance, equal to that of standard-size nebulizers. Ask your pharmacist to stock the new DeVilbiss No. 41 Pocket Nebulizer. $5.00 cost to patient. The DeVilbiss Company, Somerset, Pa., and Barrie, Ontario.

DeVILBISS ATOMIZERS NEBULIZERS VAPORIZERS

SOMERSET, PA.

"The Line the Physician Knows and Prescribes"

The DeVilbiss Company Dep't. P
Somerset, Pa.

Enclosed is $1.00 for DeVilbiss No. 41 Pocket Nebulizer to be used as an office demonstrator. A special introductory offer limited to the medical profession.

NAME....., M. D.

STREET.....

CITY.....STATE.....

(Not valid after July 1, 1954)

Supplement your experience with balanced coverage of a broad field . . .

READ

A.M.A. ARCHIVES OF PATHOLOGY

SIGNIFICANT DEVELOPMENTS BROUGHT TO YOU AS THEY OCCUR . . .

Original investigations based either on morphologic study and clinical-pathologic correlation or on results from experimental work . . .

Special articles covering topics of interest to anatomic or clinical pathologists or describing new or modified techniques . . .

Case reports based on a new or unfamiliar entity . . . a new feature of an established disease . . . additional examples of a rare disease whose nature is not clearly established . . .

$8.00 YEARLY $8.50 CANADIAN
$9.25 FOREIGN

You will want to keep informed on this expanding field. Write to the AMERICAN MEDICAL ASSOCIATION, 535 North Dearborn Street, Chicago 10, Illinois, for your monthly copy of A.M.A. Archives of PATHOLOGY

NOW!

A NEW GOOD-LITE!

CHECK THESE NEW FEATURES

- New! "2 way" transformer with primary on-off switch and 2 positions—Bright and Ultra Bright.
- New! Long Life bulbs give more even field of light.
- New! Form fitting headbands in Polyethelene plastic, glass clear plastic, white buterate plastic, leather or fibre. Your choice of soft leather or sponge rubber pads.
- The Good-Lite Headlight employs one of the most powerful lighting arrangements ever built into this type of equipment. 750 foot candles of light can be directed exactly where you want it. Completely adjustable . . . the Good-Lite can be used over one eye for deep narrow cavities, between the eyes for shallow cavity work, or at the side for oblique illumination.

GOOD-LITE UNIT INCLUDES

- Good-Lite Parallel Headlight

Two interchangeable mirrors

Two extra long life bulbs

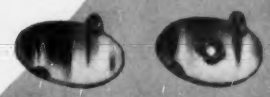
New "2 Way" Shock-proof rubber connections

Choice of 4 types of headbands

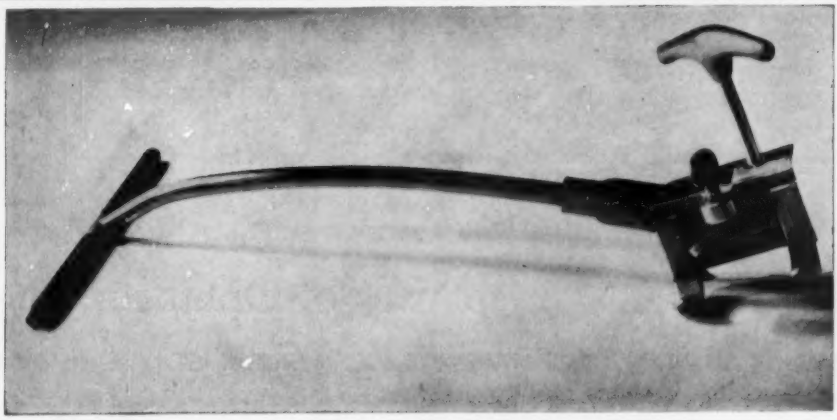
Consult your surgical dealer or write today for catalog and prices on the New Good-Lite.

TWIN MIRRORS ARE READILY INTERCHANGEABLE

Twin Good-Lite Mirrors provide two types of light-center beam light, for deep cavity surgery; plain mirror light, for shallow cavity surgery. By removing mirror, you get direct light for surface surgery.



GOOD-LITE CO.
7638 MADISON STREET
FOREST PARK, ILLINOIS

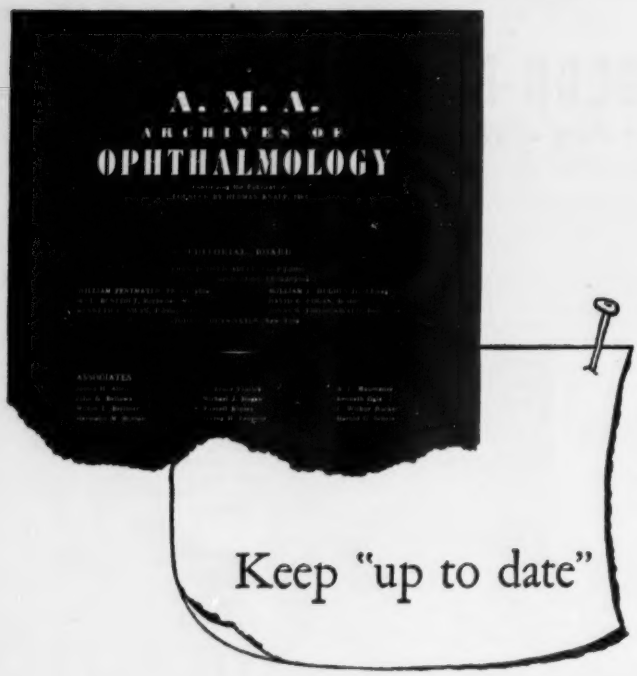


The New and Improved LEWY LARYNGOSCOPE HOLDER

An external chest support with unusual power for obtaining easy and adequate exposure with standard laryngoscopes through its leverage, this useful new instrument (with Doctor Lewy's technique) is described in ARMAMENTARIUM, Volume II, Number 1. If you have not already received your copy of this publication, we'll send it to you promptly upon request.

V. Mueller & Co.

Instrument Makers to the Profession Since 1895
330 S. Honore Street, CHICAGO 12, ILLINOIS



A timely publication on the latest developments and techniques of Ophthalmology.

Read clinical reports, original papers by foremost authorities . . . book reviews, abstracts, news and comment . . .

A special journal for the medical profession covering diseases of the eye, its relation to general health, techniques and corrections . . .

A.M.A. archives of Ophthalmology

AMERICAN MEDICAL ASSOCIATION

535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

Please enter a subscription to A.M.A. archives of OPHTHALMOLOGY for one year.

☐ I enclose $..... ☐ Please bill me

NAME.....

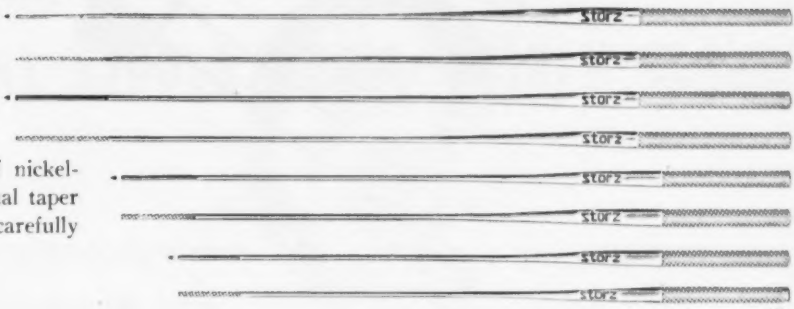
ADDRESS.....

CITY..... ZONE..... STATE.....

$12.00 YEARLY **$13.50 FOREIGN** **$12.50 CANADIAN**

storz Surgical Instruments

Better Applicators



Uniformly made of nickeloid, having a gradual taper which avoids kinks, carefully finished tips.

Storz Instruments are not sold under any other Trade Mark . . . Available thru our organization only.

Catalog upon request

		Doz.
5445	Applicator, Ear, LATHBURY: rough-twist end, regular weight, 6″	$5.50
5446	Applicator, Ear, LATHBURY: triangular end, regular weight, 6″	5.50
5447	Applicator, Ear, LATHBURY: rough-twist end, light weight, 6″	5.50
5448	Applicator, Ear, LATHBURY: triangular end, light weight, 6″	5.50
5450	Applicator, Nasal, UEBE: rough-twist end, regular weight, 7″	6.00
5451	Applicator, Nasal, UEBE: triangular end, regular weight, 7″	6.00
5452	Applicator, Nasal, UEBE: rough-twist end, light weight, 7″	6.00
5453	Applicator, Nasal, UEBE: triangular end, light weight, 7″	6.00
5455	Applicator, Sinus, LUDWIG: rough-twist end, delicate, 7″	6.50
5456	Applicator, Sinus, LUDWIG: triangular end, delicate, 7″	6.50

Storz Instrument Company

4570 AUDUBON AVENUE ST. LOUIS 10, MO.

In acute and chronic

Sinusitis

By using the NICHOLS NASAL SYPHON, these cavities may be safely and successfully drained of mucopurulent material, thus affording prompt symptomatic relief. Used and prescribed for more than 32 years. Prove its effectiveness on your most troublesome cases. Just mail the coupon.

Nichols NASAL SYPHON

NICHOLS NASAL SYPHON, INC. MONTCLAIR, N. J.

Please send details of SPECIAL OFFER "S" without obligation.

NAME.....M.D.

ADDRESS.....

CITY.....ZONE.....STATE.....

prompt antibacterial and deodorizing action in otitis

Furacin® Anhydrous Ear Solution

brand of nitrofurazone, Eaton

Furacin greatly simplifies the treatment of otitis media and externa because it combines significant advantages. Its antibacterial action extends to the great majority of clinically important pathogens concerned. Cure or marked improvement is rapid in the majority of patients.[1,2] Patients with chronic otitis media will be grateful for the marked diminution of the malodorous discharge.[3]

(1) Douglass, C. C.: Laryngoscope 58: 1274, 1948; (2) Anderson, J. R., and Steele, C. H.: Laryngoscope 58: 1279, 1948; (3) Long, P. H.: A-B-C's of Sulfonamide and Antibiotic Therapy, Philadelphia, W. B. Saunders, 1948, p. 152.

EFFECTIVE IN THE PRESENCE OF PUS • MIXES READILY WITH EXUDATES • VIRTUALLY NONIRRITATING • NEGLIGIBLE TOXICITY FOR HUMAN TISSUES • STABLE

Formula: Furacin Anhydrous Ear Solution contains Furacin 0.2% dissolved in a nonaqueous, hygroscopic liquid vehicle composed of polyethylene glycol.

Supplied: Dropper bottle of 30 cc. (1 fl. oz.)

EATON LABORATORIES
NORWICH, NEW YORK

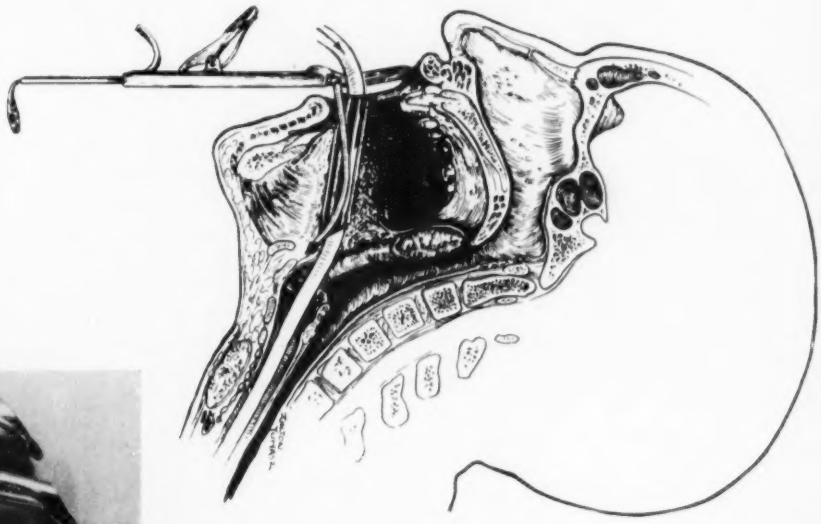
ACCEPTED COUNCIL ON PHARMACY AND CHEMISTRY AMERICAN MEDICAL ASSOCIATION

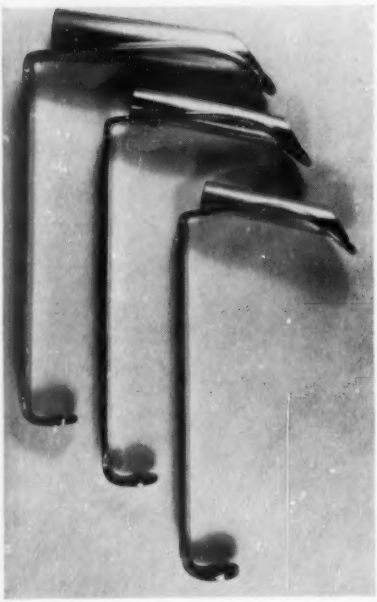
THE NITROFURANS—A UNIQUE CLASS OF ANTIMICROBIALS o.u.p. PRODUCTS OF EATON RESEARCH

Now Available

THE BARTON ENDOTRACHEAL TONGUE BLADE*

For Modern Oro-Pharyngeal Surgery





THE BARTON ENDOTRACHEAL TONGUE BLADE provides:

1. An unobstructed airway avoiding hypoxia.
2. A more pleasant general anesthetic.
3. No interference with surgical field.
4. Complete protection against tracheobronchial aspiration.

THE BARTON ENDOTRACHEAL TONGUE BLADE is manufactured in three sizes to fit any patient from infant to adult. . . . Sizes 28F, 30F, 42F. All blades fit the Crowe-Davis or similar mouth-gags. Set of three $40, Individually $15.

*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

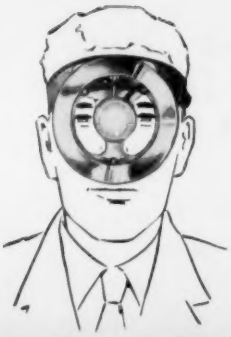
A QUALITY INSTRUMENT OF AIRCRAFT STAINLESS STEEL
ORDER BARTON ENDOTRACHEAL TONGUE BLADES FROM:

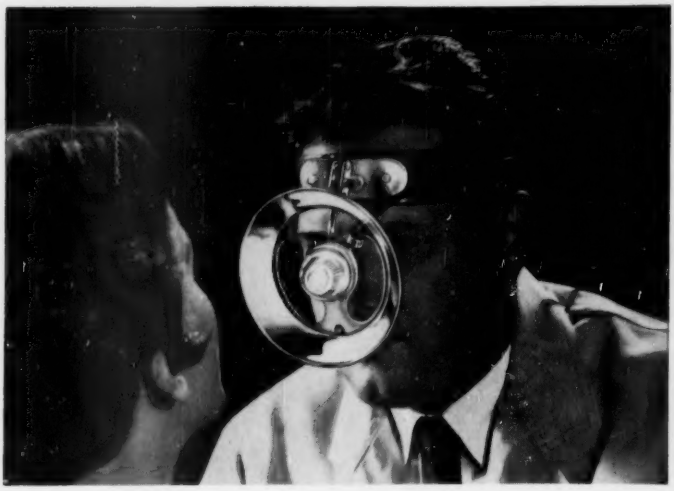
The Broton-Hiltra Company

Box 11324, Briggs Station **Los Angeles 48, California**

You may look like a man from Mars, Doctor . . .

but you'll see exactly what you're doing





WELCH ALLYN *Full Beam* HEADLIGHT

U.S. Patent No. 2,651,301

BINOCULAR VISION THROUGH THE BEAM OF LIGHT

Normal depth perception is retained and visible shadows in the instrumentation area are eliminated. Can be swung instantly to forehead height for oblique lighting.

GREATER INTENSITY AND EVENNESS OF ILLUMINATION

450 foot candles for illuminating deepest body cavities, yet glare and specular reflection are completely absent.

DURABLE, PRACTICAL, ECONOMICAL

Nothing to break or get out of adjustment. The bulb is a standard 1000 hour automobile taillight lamp. Complete with transformer for 110-120 volt, 60 cycle, AC current.

No. 450 Headlight with transformer $36.00

WELCH ALLYN, INC., SKANEATELES FALLS, NEW YORK

DESIGNERS AND MANUFACTURERS OF DIAGNOSTIC INSTRUMENTS FOR 39 YEARS